A. LIKHACHOV

DISEASES OF THE EAR, NOSE AND THROAT

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By A. G. Likhachov, M. D.

Professor of Otorhinolaryngology and Director of the Clinic of Otorhinolaryngology of the Moscow First Medical Institute; Chief Otorhinolaryngologist of the U.S.S.R. Ministry of Public Health; President of the U.S.S.R. Otorhinolaryngological Society

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Проф. А. Г. ЛИХАЧЕВ

БОЛЕЗНИ УХА, ГОРЛА и НОСА

CONTENTS

DISEASES OF THE NOSE, PHARYNX AND LARYNX

PREFACE

Since the end of the past century, diseases of the ear, nose and throat have been regarded as a single subject because of the anatomic proximity of these organs and the interdependence of their affections. Another reason is that these organs have to be examined by endoscopic methods requiring artificial lighting and appropriate instruments.

Otorhinolaryngology is the science of the diseases of the ear, nose and throat (E.N.T.). It is one of the young branches of medicine that have been extensively developed in the Soviet Union only since the Great October Socialist Revolution.

In pre-revolutionary Russia, otorhinolaryngology was still in its infancy, and specialised medical aid was available only to a small minority of the people. After the Great October Socialist Revolution, nation-wide efforts were made to provide free, as well as specialised medical aid to the population. Otorhinolaryngology was widely developed: an extensive network of out- and in-patient E.N.T. clinics was set up to ensure skilled medical attention for the public. The proper organisation of otorhinolaryngological aid, and the broad practical use of the latest methods of treatment have considerably reduced mortality from these diseases in the U.S.S.R.

The prophylactic aspects of otorhinolaryngology have also been given great attention in Soviet times. The importance of prophylaxis of the upper respiratory tract is evidenced not only by a decrease in disease incidence, but by the improved physical development, particularly of children.

The prerequisites for successful and active prevention and treatment of E.N.T. diseases include a further practical application and creative development of the concepts of

nervism advanced by I. M. Sechenov and S. P. Botkin and elaborated by I. P. Pavlov and his followers. These concepts have come to be a guiding principle in clinical practice.

The principle of Soviet medicine—to cure the patient rather than the disease—underlies otorhinolaryngological practice in the U.S.S.R. and permits of a correct methodological approach to the therapy of ear, nose and throat diseases.

The present text-book of otorhinolaryngology is intended for secondary medical schools and gives the most essential theoretical and practical information needed by the junior medical personnel engaged in independent practice.

It is calculated to enable the junior medical personnel, employed as assistant physicians in medical institutions or working on their own, to diagnose typical diseases of the ear, nose and throat, prescribe and give correct treatment, and if need be, render first aid to the patient.

Before discussing the clinical aspects of diseases of the ear, nose and throat, we deem it necessary to give a concise description of the anatomy and physiology of these organs, which should considerably facilitate the clinical study.

Special attention is devoted to early diagnosis of ear, nose and throat diseases, which is very important for both treatment and prophylaxis.

The pathogenesis, symptomatology and course of major diseases (acute otitis, acute mastoiditis, aggravated chronic suppurative otitis, labyrinthitis, otogenic meningitis, otogenic sepsis, otogenic brain abscess, frontal sinusitis, quinsy, laryngitis, etc.) are discussed in greater detail to help assess the morbid condition in each particular case and, if necessary, to refer the patient in due time either to a hospital or to a specialist for consultation.

In describing the various diseases the author has endeavoured to lay stress upon the prophylactic trend in otorhinolaryngology by pointing out the value and methods of individual prophylaxis, body hardening, etc.

In January 1960, the Central Committee of the Communist Party of the Soviet Union and the U.S.S.R. Council of Ministers passed a decision on "Measures to Improve Medical

Care and Public Health in the U.S.S.R.", which is a broad programme of further development of the public health services.

This nation-wide introduction of health measures can be achieved only with the aid of the army of the junior medical workers which is more than one million strong. As a clinical subject otorhinolaryngology not only helps in early diagnosis of many infectious diseases, but also plays an equally important role in preventing their complications.

Another important problem today is that of treating severe diseases and their aftereffects as a method of reducing and restoring to health the still numerous contingent of patients with chronic suppurative otitis media and chronic diseases of paranasal sinuses and the pharynx (chronic tonsillitis, etc.).

The efforts of junior medical personnel are an invaluable contribution to the solution of these and other practical health problems, which fact lends particular importance to the training of qualified junior medical personnel.

In view of the singular importance of prophylaxis in reducing the incidence of diseases and preventing serious complications of certain ear, nose and throat diseases, some chapters (on chronic suppurative otitis media, burns of the pharynx and esophagus, scleroma, etc.) have sections on prophylaxis. The sections dealing with treatment of some diseases (scleroma, ozena, esophageal burns, etc.) are supplied with modern data, especially on antibiotics.

In view of the importance of health education in the campaign for prophylaxis of the upper respiratory tract and prevention of deafness and amblyacousia (dullness of hearing), a list of recommended topics for lectures in health education with a schematic description of two such lectures is offered at the end of the book.

The author expresses the hope that the present edition compiled with due regard for all the criticisms and useful suggestions received will prove helpful in the training of junior medical personnel.

A. G. Likhachov

DISEASES OF THE EAR

ANATOMY OF THE EAR

The ear consists of three parts—the external, the middle and the internal (Fig. 1), with the latter containing the receptors of the auditory and vestibular analysors.

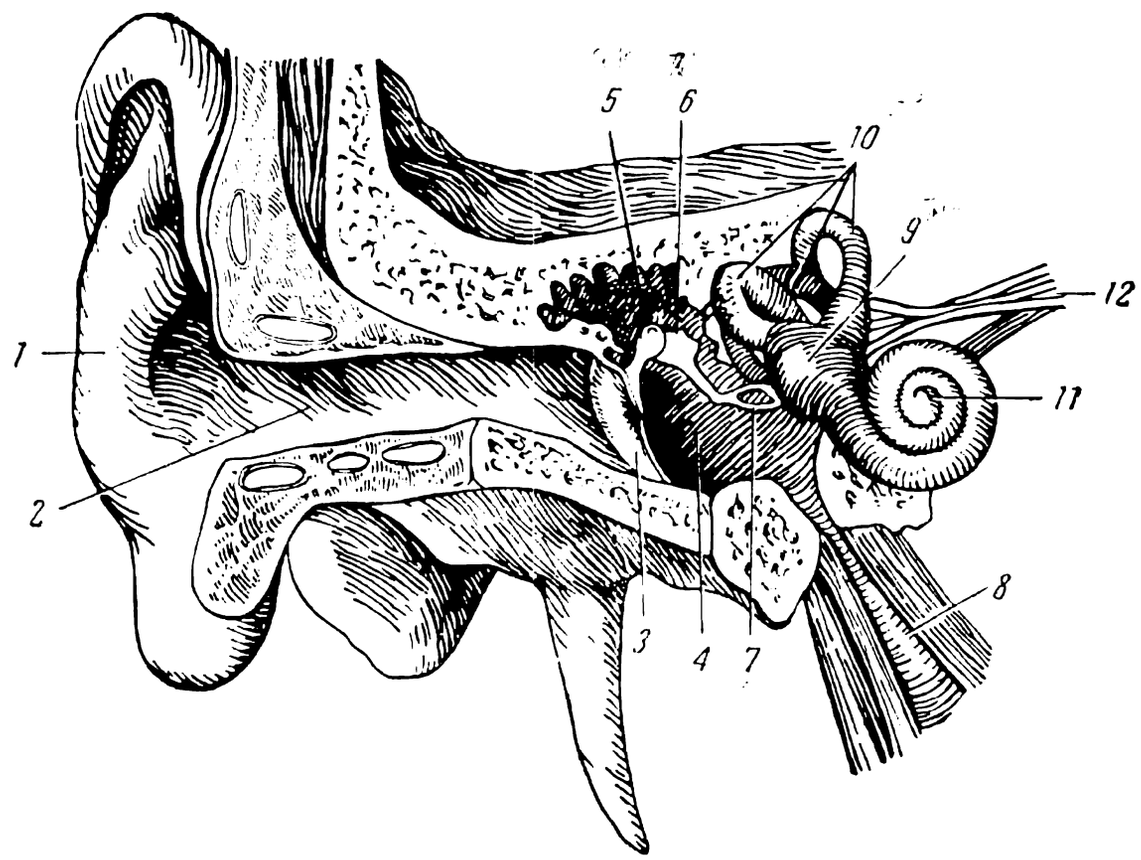


Fig. 1. Anatomy of the External, Middle and Internal Ear (Semi-Schematic View)

(1) pinna; (2) external auditory meatus; (3) tympanic membrane; (4) tympanic cavity; (5) malleus; (6) incus; (7) stapes; (8) Eustachian tube; (9) vestibule; (10) semicircular canals; (11) cochlea; (12) auditory (acoustic) nerve

External Ear

The external ear comprises the pinna, or auricle, and the external auditory meatus.

The shell-shaped *pinna* (Fig. 2) is composed of a skin-covered cartilaginous lamella whose posterior surface is evenly convex and smooth, while its anterior surface is concave, with semilunar folds and hollows between them. The skin on the anterior surface of the pinna adheres directly to the perichondrium; on the posterior surface, however, it may form folds owing to the presence of a small layer of loose cellular tissue. The free anteroexternal margin of the pinna is known as the *helix*; towards the bottom the pinna gradually turns into the *lobe* devoid of cartilage and consisting of well-developed fat and cellular tissue with a small number of vessels and nerves. The small protuberance of cartilage projecting over the external auditory meatus is named the *tragus*. In front of the helix and parallel to it is a ridge known as the *anthelix*, with the *antitragus* at its posterior end.

The *external auditory meatus* extends from the funnel-shaped hollow (*cavum conchae*) on the outer surface of the pinna to the tympanic membrane or drum. It is a canal directed horizontally inwards and a little forward. Its average length from the tragus top to the drum edge is 3.5 cm. The drum at the end of the canal separates the external and the middle ears. The outer third of the auditory canal consists of cartilage and membranous tissue, and both inner portions of bone.

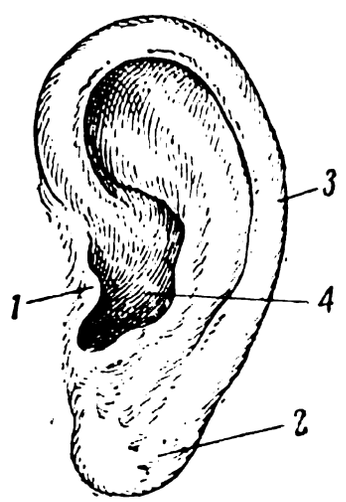


Fig. 2. Pinna

(1) tragus; (2) lobe; (3) helix; (4) cavum conchae

The external auditory meatus is curved in the horizontal and frontal planes. The cartilaginous and bony portions of the meatus form an obtuse angle opening forward and downwards. Therefore, when examining the drum, the pinna must be pulled backwards and upwards, in order to straighten out the meatus. The oval lumen of the external auditory meatus has a longitudinal diameter of 1 cm. Its width varies with age and in

different individuals. Its narrowest part is the isthmus, where the cartilaginous and bony portions form a junction and where foreign bodies are most likely to lodge. The walls of the auditory meatus are lined with skin which in the bony portion gradually becomes thinner, loses its subcutaneous tissue and accretes closely with the periosteum. The skin covering the cartilaginous portion abounds in hair, sebaceous glands and ceruminous glands which secrete the earwax, or cerumen. The skin of the bony portion has neither hair, nor glands. The external bony meatus has four walls: the *superior* wall formed by the squamous portion of the temporal bone, its internal part bordering on the floor of the middle cranial fossa; the *posterior* wall serving as the front wall of the mastoid process; the *anterior* and *inferior* walls whose inner parts are formed by the tympanic portion of the temporal bone. The external third of the anteroinferior wall is made up of cartilage with two vertical fissures through which an inflammatory process in the external auditory meatus can spread to the connective tissue surrounding the parotid gland, and vice versa. The anterior wall adjoins the articular head of the mandible, which explains why it is painful to open the mouth and chew in cases of inflammation of the anterior wall of the external auditory meatus. Injury to the lower jaw, a fall, or an upward blow to the chin may cause a fracture in the anterior wall of the auditory meatus with the articular head of the mandible pushed backwards and upwards.

In the newborn, there is neither bony auditory meatus nor mastoid process, and in place of the former there is a bony ring or *annulus*, which is deficient in a small upper section, and is directly connected with the membranocartilaginous auditory meatus. The inner border of the annulus has a bony furrow (sulcus) into which the tympanic membrane is inserted. In the bone-free upper part, the drum is directly attached to the lower edge of the squamosa, the so-called *notch of Rivinus*. By the end of the third year the external auditory meatus is fully developed.

The external ear is supplied with blood by branches of the external carotid artery. It is innervated, in addition to the trigeminal branches, by the auricular nerve (*ramus*

auricularis n. vagi) ramified in the posterior wall of the auditory meatus. Mechanical irritation of the latter wall, as in wax removal, often causes reflex cough. The lymph from the walls of the auditory meatus drains into the nearest lymph nodes located in front of the auricle, on the mastoid process, and under the inferior wall of the auditory meatus. Inflammations in the external auditory meatus are often accompanied by swelling and pain in these lymph nodes.

Tympanic Membrane

The tympanic membrane (Fig. 3, coloured Table I, Fig.1) or drum is a thin semi-transparent elliptical disc situated between the external and middle ears. It is 9.5-10 mm × 8.5-9 mm in size. The greater part of the drum fitted into the bony furrow of the tympanic ring is taut, and is called the *pars tensa*; the other, smaller part of the drum facing forward and upwards and directly attached to the incisure in the squamosa known as the notch of Rivinus (*incisura Rivini*) is lax, and is called the *pars flaccida* or *Shrapnell's membrane*. The drum consists of three layers: an outer or

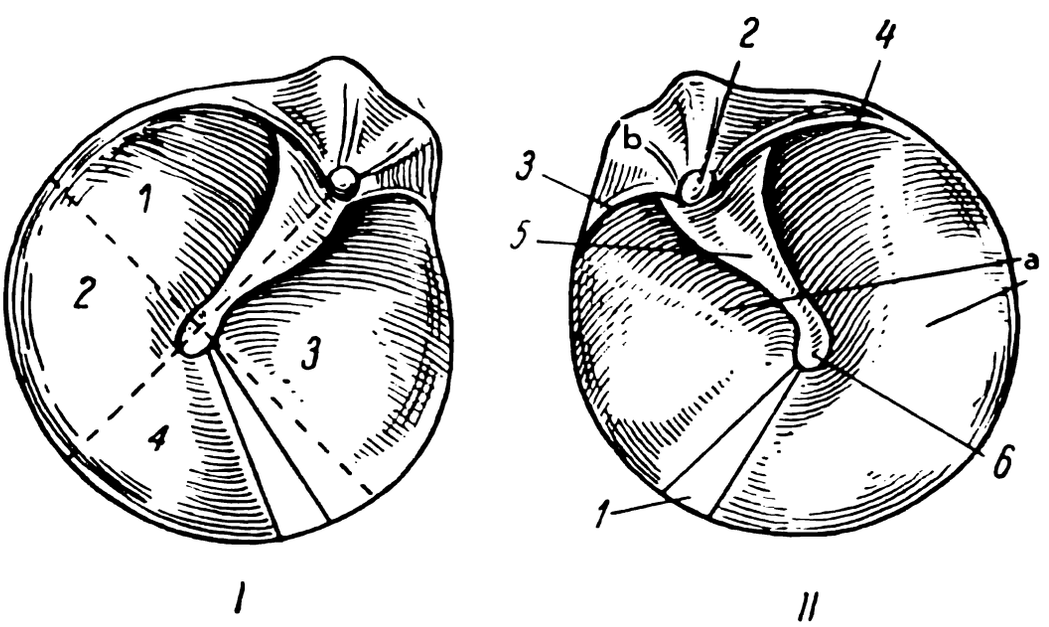


Fig. 3. Normal Drum. Right and Left Sides

I—right drum is divided into four quadrants: (1) posterosuperior; (2) posteroinferior; (3) anterosuperior; (4) anteroinferior.
II—left drum: (a) pars tensa; (b) pars flaccida or Shrapnell's membrane; (1) light reflex (cone); (2) short process of malleus; (3), (4) anterior and posterior folds; (5) handle of malleus; (6) umbo

epidermal layer continuous with that of the auditory meatus, a middle layer of radiating and circular connective tissue fibres, and an inner layer of mucosa continuous with the mucous membrane of the tympanic cavity. Shrapnell's membrane or pars flaccida consists only of two layers and lacks the middle stratum of fibrous tissue.

In early childhood, the drum is comparatively thick owing to the presence of a loose submucous layer. It grows compact with time and in old age becomes quite thin.

The drum is placed obliquely and not perpendicularly to the long axis of the auditory meatus, so that it faces forward, downwards and inwards. In the newborns and breast-fed babies, the drum is almost horizontal.

Examination of the drum through the auditory meatus reveals a funnel-shaped concavity in its centre with an eminence called the *umbo* in its deepest place. The handle of the malleus embedded in the fibrous layer of the drum starts from the umbo and goes forward and upwards to end above in a tiny knob the size of a pin-head—the short process. The two folds stretching anteriorly and posteriorly from the short process separate the upper lax membrana flaccida from the lower taut membrana tensa.

Middle Ear

The middle ear comprises the tympanic cavity, the mastoid process with its cellular system and the Eustachian tube (Fig. 4), all directly interconnected.

The *tympanic cavity* is a small chamber, about 1 cu cm in size, lying in the depth of the temporal bone, between the tympanic membrane and the internal ear. In front, through the Eustachian tube, the tympanic cavity communicates with the nasopharynx; behind, through the entrance into the mastoid antrum (*aditus ad antrum mastoideum*), it communicates with the latter and the cells of the mastoid process. The tympanic cavity, similar to the cells of the mastoid process, contains air coming through the Eustachian tube.

It is customary to divide the tympanic cavity into three parts: the middle and biggest part, *mesotympanum*, corresponding to the pars tensa of the drum; the upper part, *epitympanum*, lying above the former and also known as the

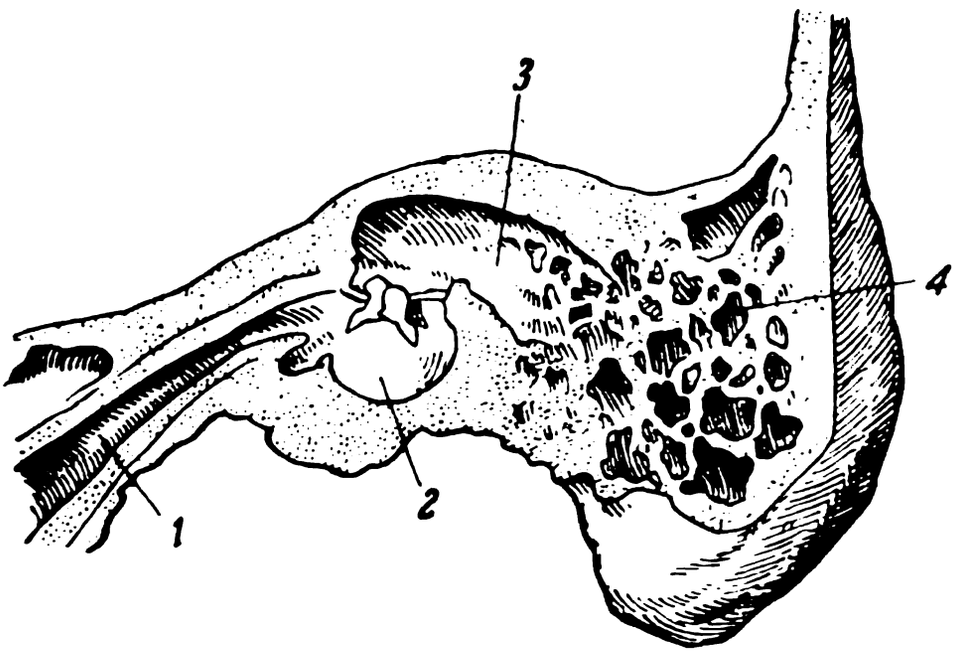


Fig. 4. Section Showing Middle Ear

(1) Eustachian tube; (2) tympanic cavity; (3) antrum; (4) mastoid cells

epitympanic recess or attic; the lower part, *hypotympanum*, lying below the drum level.

The tympanic cavity has six walls.

The *roof* of the tympanic cavity is a thin plate of bone separating the tympanic cavity from the middle cranial fossa where the temporal lobe is situated. This plate often has congenital fissures through which vessels pass from the middle cranial fossa. These anatomical features may account for the meningeal symptoms frequently observed in young children with acute otitis media.

The *inferior wall* or floor of the tympanic cavity is separated from the jugular bulb by a fairly thick bony plate. Bone fissures in this wall are rarely found.

The Eustachian tube begins with an opening in the *anterior wall* separating the tympanic cavity from the internal carotid canal.

An opening in the upper part of the *posterior wall* leads to the mastoid antrum (*aditus ad antrum mastoideum*).

The *internal wall* separates the tympanic cavity from the internal ear. It is marked by a gentle eminence, the promontory (*promontorium*), corresponding to the basal turn of the cochlea. Above and behind the promontory is an oval window or the fenestra vestibuli which leads into the vesti-

bule and is closed by the foot plate of the stapes. Behind and below the promontory in a niche is a round window or the fenestra cochleae which leads into the cochlea, and is filled with a thin membrane, the secondary tympanic membrane.

On the internal wall above the oval window is a bony torus—the horizontal part of the facial nerve canal. On reaching the entrance to the antrum, the facial nerve canal turns downwards to form a descending knee, then passes behind the posterior wall of the auditory meatus and through the stylomastoid foramen to the base of the skull. The walls of this bony canal may be eroded; in such cases, the middle ear mucosa may come through fissures into direct contact with the sheath of the facial nerve. This sometimes causes the development of facial paresis and paralysis in suppurative otitis media. Somewhat behind and above the facial nerve canal, on the inner wall of the aditus ad antrum, lies the peak of the horizontal semicircular canal the clear contour of which serves for orientation in operations on the middle ear.

The *external wall* of the tympanic cavity is formed by the tympanic membrane, and above the drum—by the external bony wall of the epitympanic recess or attic.

The tympanic cavity contains the three auditory ossicles—the malleus, the incus and the stapes (Fig. 5) — which are interconnected by joints and ligaments to form a continuous and rather flexible chain between the drum and the oval window. The handle of the malleus is woven, as it were, into the fibrous layer of the tympanic membrane, and the foot plate of the stapes is fixed in the oval window by means of an annular ligament. The incus lies between the malleus and the stapes. The whole system is kept in place by ligaments fastening the malleus and incus to the walls of the tympanic cavity.

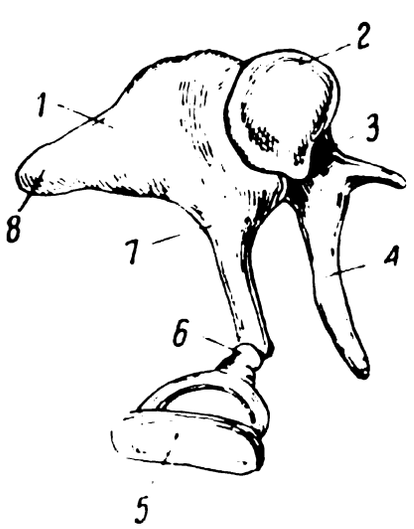


Fig. 5. Interconnected Auditory Ossicles

(1) body of incus; (2) head of malleus; (3) neck of malleus; (4) handle; (5) plate of stapes; (6) head of stapes; (7) long process of incus; (8) short process of incus

The *tympanic muscles.* There are two muscles in the tympanic cavity: 1. The *tensor tympani* muscle which stretches the tympanic membrane. It lies in the bony canal above the Eustachian tube, and is attached to the handle of the malleus. 2. The *stapedius* muscle which arises from the posterior wall of the tympanic cavity and is attached to the head of the stapes by a slender tendon. The tensor tympani is innervated by a branch of the trigeminal nerve, and the stapedius muscle by a branch of the facial nerve.

The *Eustachian* or *auditory tube* which is about 3.5 cm in length connects the tympanic cavity with the nasopharynx. The upper third of this tube, adjoining the tympanic cavity, has bony walls, while the remaining lower portion leading into the nasopharynx is made up of membrane and cartilage. The movement of the cilia of the ciliated epithelium lining the Eustachian tube is towards the nasopharynx. At rest, the Eustachian tube is in a collapsed state, but with each swallowing movement it opens by contraction of the soft palatal muscles attached to it, to let air into the tympanic cavity.

The *mastoid process* located just behind the external auditory meatus is a bony structure protruding downwards with the sternocleidomastoid muscle attached to it. In young children, the mastoid process is not fully developed and represents a bony tubercle behind the osseous tympanic ring.

The upper border of the process is the temporal line (*linea temporalis*), a bony torus which is a backward extension of the zygomatic process. The floor of the middle cranial fossa usually lies on a level with this line.

The *anterior wall* of the mastoid process is the posterior bony wall of the external auditory meatus. Behind the spot where the superior wall of the auditory meatus merges with the posterior wall, there is a small bony peak or the suprameatal spine (*spina supra meatum*) lying above the external auditory meatus. Behind the spine there is a smooth depression, the mastoid fossa (*fossa mastoidea*). The suprameatal spine and the temporal line are important landmarks in surgical operations; the mastoid antrum (*antrum mastoideum*) lies on the projection of the mastoid fossa (*fossa mastoidea*) in the depth of the mastoid process.

The *internal wall* of the mastoid process abuts upon the

labyrinth, and more posteriorly is bordered by the post-cranial fossa. On the surface facing the post-cranial fossa there is a rather wide S-shaped groove, the sigmoid sulcus, containing part of the sigmoid sinus of the dura mater. The central part of the mastoid process is the antrum lying just behind the epitympanic recess. The antrum communicates with the tympanic cavity and the air-filled cells of the mastoid process. The superior wall or roof of the antrum separates it from the middle cranial fossa.

The following types of structure are to be found in the mastoid process: the pneumatic or large-celled, the diploic and the compact or "sclerotic". In the case of pneumatic structures, the cavity of the mastoid process is divided by thin bony partitions into a lattice of larger and smaller cells. The diploic structure has tiny cells resembling a diploetic bone; the most frequent is the mixed form of mastoid structure where smaller cells are to be found alongside bigger ones. In compact structures the bone is indurated and the cells are very few; this structure frequently occurs as a result of chronic suppurative otitis media.

The walls of the tympanic cavity, antrum and mastoid cells are lined with a continuous thin mucosa devoid of mucous glands. The mucous membrane of the Eustachian tube and of the adjoining part of the tympanic cavity floor is covered with ciliated columnar epithelium; the mucosa of the cartilaginous part of the Eustachian tube contains mucous glands which are absent in the mucosa of the other parts of the middle ear.

The middle ear is supplied with blood mainly by branches of the external carotid artery. Venous blood drainage from the middle ear is maintained by the veins of the dura mater, the venous sinuses and the venous plexuses round the carotid artery. Lymph drainage is carried out in two ways: 1. through the lymphatic vessels of the Eustachian tube to the retropharyngeal lymph nodes and further to the deep cervical glands; 2. through the lymphatic vessels across the tympanic cavity to the lymphatic ducts of the external auditory meatus and the lymph nodes in front of and behind the auricle. The nerve supply of the middle ear is through branches of the glossopharyngeal, facial and sympathetic nerves.

Internal Ear or Labyrinth

The internal ear consists of membranous and bony labyrinths, the latter surrounding the former like a capsule. The membranous labyrinth is filled with fluid known as endolymph, while around it and separating it from the bony shell is the spinal fluid known as perilymph.

The bony labyrinth is made up of the vestibule, three semicircular canals and the cochlea (Fig. 6).

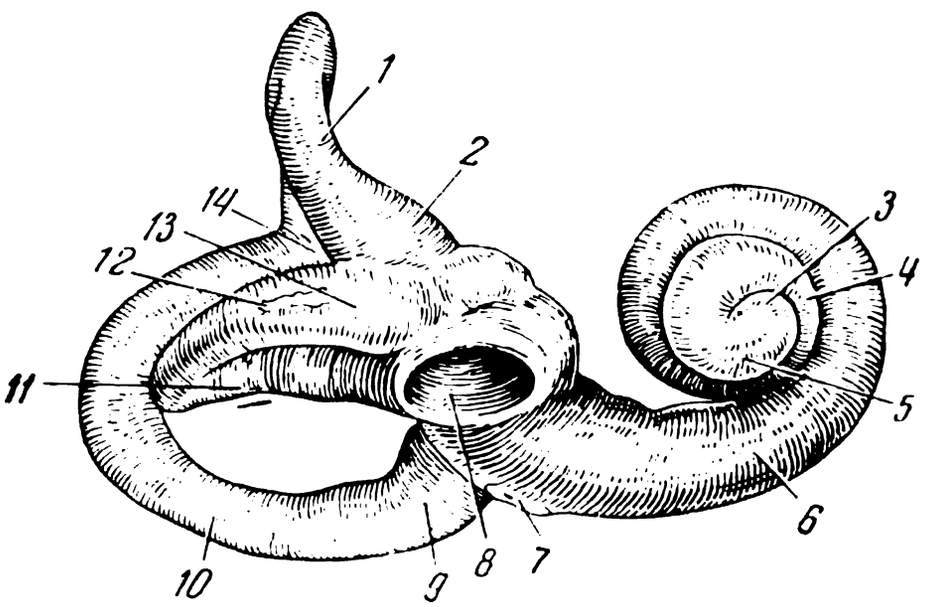


Fig. 6. Bony Labyrinth on Right Side

(1) frontal semicircular canal; (2) ampulla of frontal semicircular canal; (3) apex of cochlea; (4) medial turn of cochlea; (5) apical turn of cochlea; (6) basal turn of cochlea; (7) round window; (8) oval window; (9) ampulla of sagittal semicircular canal; (10) sagittal semicircular canal; (11), (12) crura; (13) ampulla of horizontal semicircular canal; (14) crus commune of frontal and sagittal semicircular canals

The *vestibule* (*vestibulum*) lies in the centre of the bony labyrinth on whose external wall is the oval window; on the opposite, internal wall there are two recesses for the two membranous sacs of the vestibule. The front sac known as the saccule (*sacculus*) communicates with the membranous cochlea lying before the vestibule, while the rear sac or utricle (*utriculus*) is connected with the three membranous semicircular canals passing behind and above the vestibule. The intercommunicating sacs of the vestibule contain the statokinetic receptors or *maculae acusticae*, otolithic or-

gans made up of a highly-differentiated specific neuroepithelium covered with a membrane containing granules of carbonate and phosphorate of lime, i.e., the otoliths.

The semicircular canals are set at right angles to each other and represent the three planes of space. They are three in number: the external or horizontal, the superior or frontal, and the posterior or sagittal. One end of each canal opens out into a larger space known as ampulla, the other end is even. The frontal and sagittal canals have a common even stem (*crus commune*).

The ampulla of each membranous canal contains a ridge, the *crista ampullaris*, which is a receptor, i.e., a nerve ending consisting of a highly-differentiated neuroepithelium or hair and supporting cells.

The free surface of the hair cells is covered with hairs which respond to the slightest displacement or pressure of the endolymph.

The receptors of the vestibule and semicircular canals are the peripheral nerve endings of the vestibular analysor.

The *cochlea* is a bony tube which describes two-and-a-half turns around a central pillar called the modiolus and resembles a snail-shell in appearance.

An osseo-membranous lamina leading from the modiolus to the external wall and also turning round the former, divides the tube lumen into two directions, the upper or scala vestibuli and the lower or scala tympani which communicate at the apex of the cochlea through a small opening known as the helicotrema. Both channels are filled with perilymph.

The scala vestibuli communicates with the vestibule, while the scala tympani borders on the tympanic cavity through the round window covered by the secondary tympanic membrane.

The scala vestibuli of the cochlea contains the thin Reissner's membrane which extends from the osseous spiral lamina to cut off a small membranous canal of triangular section filled with endolymph and known as the cochlear duct or *ductus cochlearis.*

The organ of Corti (Fig. 7), a complex receptive structure of the auditory analysor, rests on the basilar membrane (*membrana basilaris*), the lower wall of the ductus cochlea-

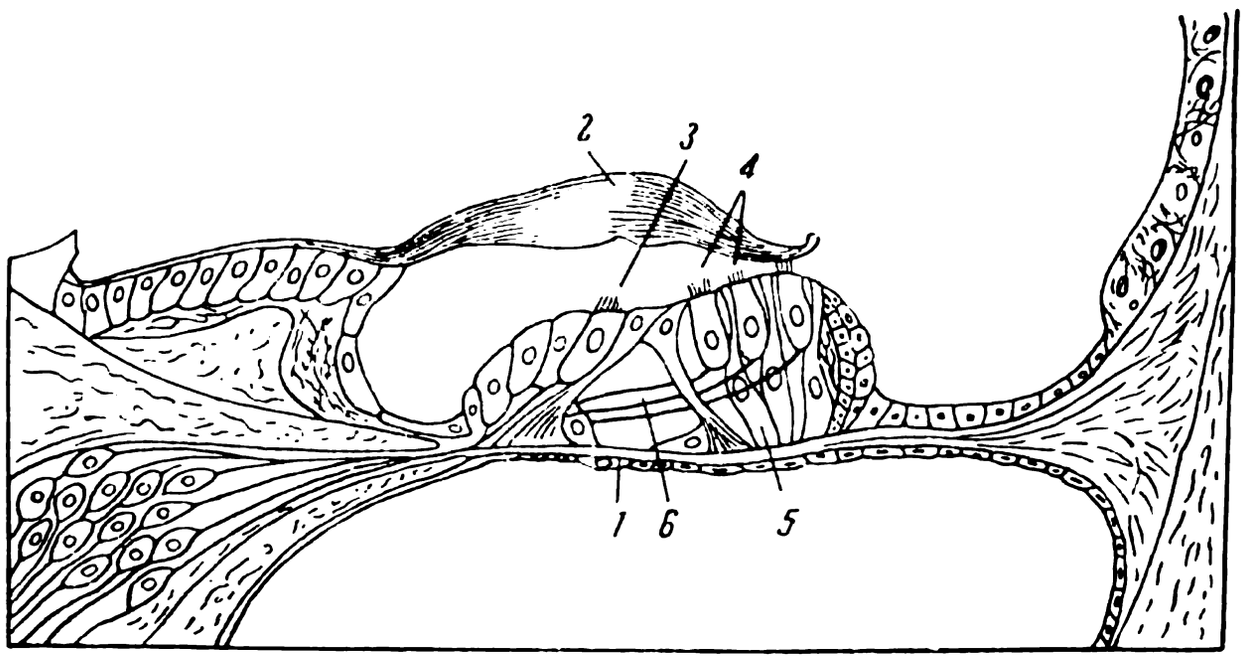


Fig. 7. Organ of Corti (Schematic View)

(1) basilar membrane; (2) tectorial membrane; (3), (4) hair cells; (5) supporting cells; (6) nerve fibres reaching hair cells

ris. The basilar membrane is an arrangement of elastic fibres of different lengths strung from the edge of the bony spiral lamina to the opposite, outer wall of the cochlea.

The organ of Corti has a very complex histological structure containing hair cells and supporting cells. The sensory cells covered with hairs are situated in small groups between the supporting cells. The cells are covered with a membrane called the tectorial membrane (*membrana tectoria*). The sensory hair cells are surrounded by a network of cochlear nerve branches leading to the spiral ganglion of the auditory nerve in the bony spiral lamina and further by intricate routes to the brain cortex.

PHYSIOLOGY OF THE EAR

The ear is one of the sense organs by which man communicates with the outer world.

I. P. Pavlov's theory of the sense organs presents them as analysors, each making up a single system of the following components: 1. the peripheral part or receptors, i.e., nerve endings adapted to respond to certain types of stimulation; 2. nerve conductors to convey the impulse from the receptor; 3. a central department in the brain cortex

for a thorough analysis of all stimuli and their transformation into sensations.

The ear performs two functions, the first is hearing, and the other is orientation in space and maintenance of equilibrium.

Auditory Function

The auditory function of the ear consists in the conduction of sounds through the external and middle ears or cranial bones and their reception by the spiral organ of Corti, the receptor of the auditory analysor. The external and middle ears make up the sound-conducting apparatus, whereas the internal ear, specifically, the organ of Corti, belongs to the sound-perceiving apparatus.

The auricle in man is of lesser importance than in animals, and yet there is no doubt that it plays a certain role in collecting sounds and determining their direction.

The external auditory meatus conducts sound waves from the outer medium to the tympanic membrane. The meatal diameter has nothing to do with hearing acuity. Its atresia, however, as well as its complete obstruction, as occurs in earwax impaction, hinders the passage of sound waves and considerably impairs the hearing.

Sound waves striking the tympanic membrane set it into vibration. The drum being connected to the handle of the malleus, these vibrations are transmitted to the ossicular chain; and the foot plate of the stapes, closing the oval window of the labyrinth, rocks in and out of the oval window according to the phase of sound vibrations. The vibration of the foot plate of the stapes in the oval window sets up vibrations in the perilymph. These vibrations are transmitted to the basilar membrane and the organ of Corti which it supports.

The vibration of the basilar membrane causes the hair cells of the spiral organ of Corti to get in touch with the overhanging tectorial membrane. At the same time, the mechanical energy of vibrations changes into the physiological process of nervous excitation which is conveyed to the most delicate receptors of the auditory nerve to be passed further to its nuclei in the medulla oblongata and through appropriate canals to the cortical auditory centres

in the temporal brain lobes where nervous impulses are interpreted as sounds heard.

The internal ear performs the most important functions of the ear, because it is here that sound perception takes place.

Normal hearing depends on the normal condition of the apparata for sound perception and conduction.

The tension of the drum and the ossicular chain necessary for normal sound conduction is maintained by the combined action of the tympanic muscles. For normal vibration the tympanic membrane requires a constant equilibrium between air pressure in the middle ear cavity and in the outer air, that is on both sides of the drum. This is maintained by the passage of air through the Eustachian tube during swallowing. Disturbance of air supply to the middle ear through the Eustachian tube causes air in the middle ear to be sucked in and the drum to be indrawn, which is followed by deterioration of hearing. The normal condition of the sound-conducting apparatus is extremely important for the transmission of low tones to the labyrinth, that is, sounds with a low frequency of vibrations per second.

There are two ways of conducting sound waves to the labyrinth: air conduction (through the external auditory meatus, the tympanic membrane and the chain of ossicles), and bone conduction (directly through the cranial bones and the stapes).

High tones, i.e., sounds of a high vibration frequency per second, are easily conducted to the labyrinth not only through the tympanic membrane and the ossicular chain, but through the cranial bones and the stapes as well.

Man can hear external sounds with a frequency of 16 to 20,000 cycles per second.

The human ear can differentiate between sounds of different pitch, intensity and timbre. There are a number of theories which seek to explain the essence of hearing and the ability of the ear to differentiate between sounds. The oldest and most widespread among them is the resonance theory advanced by Helmholtz in 1863 and based on the physical phenomenon of sympathetic vibration. According to this theory the fibres of the basilar membrane vibrate in unison with sounds, similar to the action of strings in certain

musical instruments, such as the piano or the harp. The short, thin and tighter fibres of the basilar membrane which lie in the basal turn of the cochlea vibrate in unison, i. e., resonate when stimulated by a high tone, whereas the longer, thicker and less taut fibres in the apical turn of the cochlea resonate in response to low tones.

There are a number of serious objections to the resonance theory as it oversimplifies the essence of hearing as a physiological process by describing it from the mechanical aspect alone, and fails to give a picture of the physiological properties of the auditory analysor as a whole. It should be noted, however, that the localisation of perception of high and low tones in the basal and apical cochlear turns respectively, on which the resonance theory is based, has been confirmed by experiments and clinical observations.

In opposition to the resonance theory the so-called telephonic theory of hearing asserts that the basilar membrane vibrates all over like a telephone membrane. It denies any analysis of sound being made in the peripheral receptor contained in the cochlea. This concept has been disproved by clinical practice and experimental research.

The first to prove beyond doubt that sounds of different pitch are perceived in different parts of the cochlea was L. A. Andreyev by experiments with conditioned reflexes made in I. P. Pavlov's laboratory. The experiments were made on dogs which developed conditioned reflexes of salivation to tones of low, moderate and high frequency. After the reflex had been firmly established, the cochlea on one side was completely destroyed, and the animal retained its conditioned reflex. This was followed by a selective destruction of different parts of the cochlea.

Destruction of the cochlear apex with a thin drill caused disappearance of the conditioned reflex to low-pitched sounds, whereas destruction of the cochlear base was followed by disappearance of the reflex to high-pitched sounds. These experiments have proved that an injury to the apical turn of the cochlea causes loss of low tone perception, whereas an injury to the basal turn of the cochlea is accompanied by loss of high tone perception.

Thus, according to the teachings of I. P. Pavlov and his followers the peripheral receptor of the auditory analysor

makes a primary analysis of sound by converting the latter's mechanical energy into the physiological process of nervous excitation. This, in turn, is conveyed through nerve canals to appropriate centres in the brain cortex where the nerve impulses are finally interpreted as sounds heard. I. P. Pavlov's teaching gives a clear idea of the functions of each part of the auditory analysor, thus presenting the latter's entire activity as a single physiological process.

The faculty of locating the origin of sounds, the so-called *ototopia* depends upon binaural hearing. It is largely lost in people with unilateral hearing, who have to turn their heads in various directions to locate the origin of sound. People with two healthy ears can easily determine the direction of sounds without turning their heads.

The ability to find the direction of sounds is a function of the central nervous system. If a sound comes from one side, it arrives at the ear on the other side with an insignificant delay of 0.0006 sec.

This delay makes it possible to determine the direction of sound.

Vestibular Function

Orientation of the body and its individual parts in space is made possible by co-operation of many receptors. Apart from eye-sight, the location of the body and its parts is identified through nerve endings lying in the skin, as well as in the muscles, joints and tendons, which are called proprioceptors.

In addition to the above-mentioned receptors, the cerebellum and, above all, the vestibular apparatus perform an important function in body orientation and in maintaining equilibrium at rest and in motion. The vestibular apparatus consists of the vestibule containing the otolith system and the semicircular canals with their ampullae containing the nerve endings of the vestibular analysor.

The accelerations imparted to the body during its movement in space are adequate or specific stimulants for the nerve endings of the vestibular analysor. Movements along a straight line cause displacement of the otoliths and stimulate the receptors of the otolith, or statolith, structure contained in the vestibular sacs. Angular or rotatory mo-

tions are followed by displacement of the endolymph in the semicircular canals and stimulation of receptors in the ampullae.

Stimulation of the receptors of the vestibular analysor produces a number of reflex reactions which cause a change in the tonus of some muscle bundles of the torso, extremities, neck and eyes. This, in turn, causes the whole body to change position and maintain balance.

One of the unconditioned reflexes observed in stimulation of the semicircular canals is nystagmus which consists in a rhythmic movement of the eyes in a certain direction and back, such as lateral and vertical nystagmus. Nystagmus may be observed in different positions of the eyeball, for example, in gazing straight ahead and in an extremely sidelong glance. The observation of nystagmus is used to assess the reaction of a stimulated vestibular apparatus.

The role of the vestibular apparatus becomes particularly apparent during an acute disturbance or cessation of its function, which occurs in some diseases. The patients suffer from severe static and dynamic disorders: they are unable to stand, walk and sit; they cannot co-ordinate their movements, develop spontaneous nystagmus, etc. This is accompanied by vertigo, nausea and vomiting. Three to four weeks later these symptoms subside due to compensation from the central systems. A more or less intensive reaction of the vestibular apparatus to adequate, i.e., specific, stimulation depends on the state of the central nervous system, its higher division, the brain cortex, in particular.

EXAMINATION OF THE EAR

This examination begins with collection of data on the case, followed by inspection of the ear and a functional examination of hearing. In a complex examination of the ear, nose and throat, the latter two are always the first to be dealt with.

A case history must include an account of the symptoms as described by the patient, and the circumstances under which the illness began.

In gathering this information attention should be paid to (a) ear pain and its character; (b) pus discharge; (c) de-

terioration of hearing or total deafness; (d) tinnitus; (e) dizziness. It is equally important to find out whether the patient complains of headaches, chills, etc. Among the numerous causes of ear diseases the most frequent are inflammations in the nose and throat which produce acute otitis media. Therefore, it is important to know if the ear disease in question was preceded by influenza, acute rhinitis, etc. Then general details should be obtained, i.e., information about the general state of health, past diseases, infectious ones in particular, working and housing conditions, and other facts relating to the everyday life of the patient. Examination of the ear includes an external examination and palpation of the ear and the mastoid process, as well as inspection of the external auditory meatus and the tympanic membrane (otoscopy).

Otoscopy

Examination of the external auditory meatus and the tympanic membrane, as well as the tympanic cavity when the drum is absent, requires artificial lighting and a head mirror (Fig. 8) to reflect the light into the ear and the ear speculum (Fig. 9). The best source of illumination is a 50 to 60 W frosted bulb; the head mirror is a round slightly concave mirror 8 to 9 cm in diameter with the focal length of 20 cm and a hole in the middle. In the absence of electric lighting, any source of illumination including daylight should be used. The examination is made in a sitting posture with the

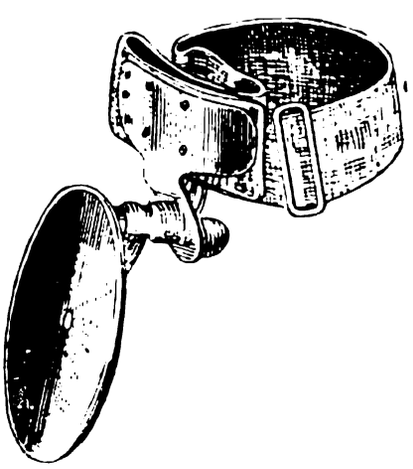


Fig. 8. Head Mirror

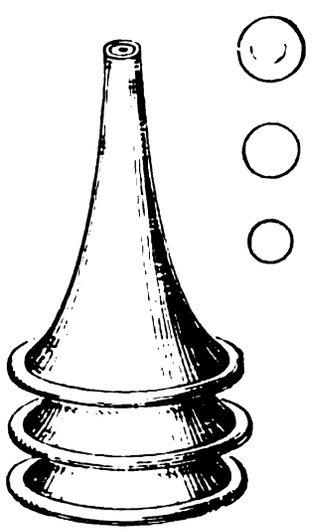


Fig. 9. Ear Speculums

light placed on a level with the patient's head, to the right and a little behind, as shown in Fig. 10. The head mirror is so fixed before the examiner's left eye that the eye, the mirror hole and the ear under examination are in one straight line.

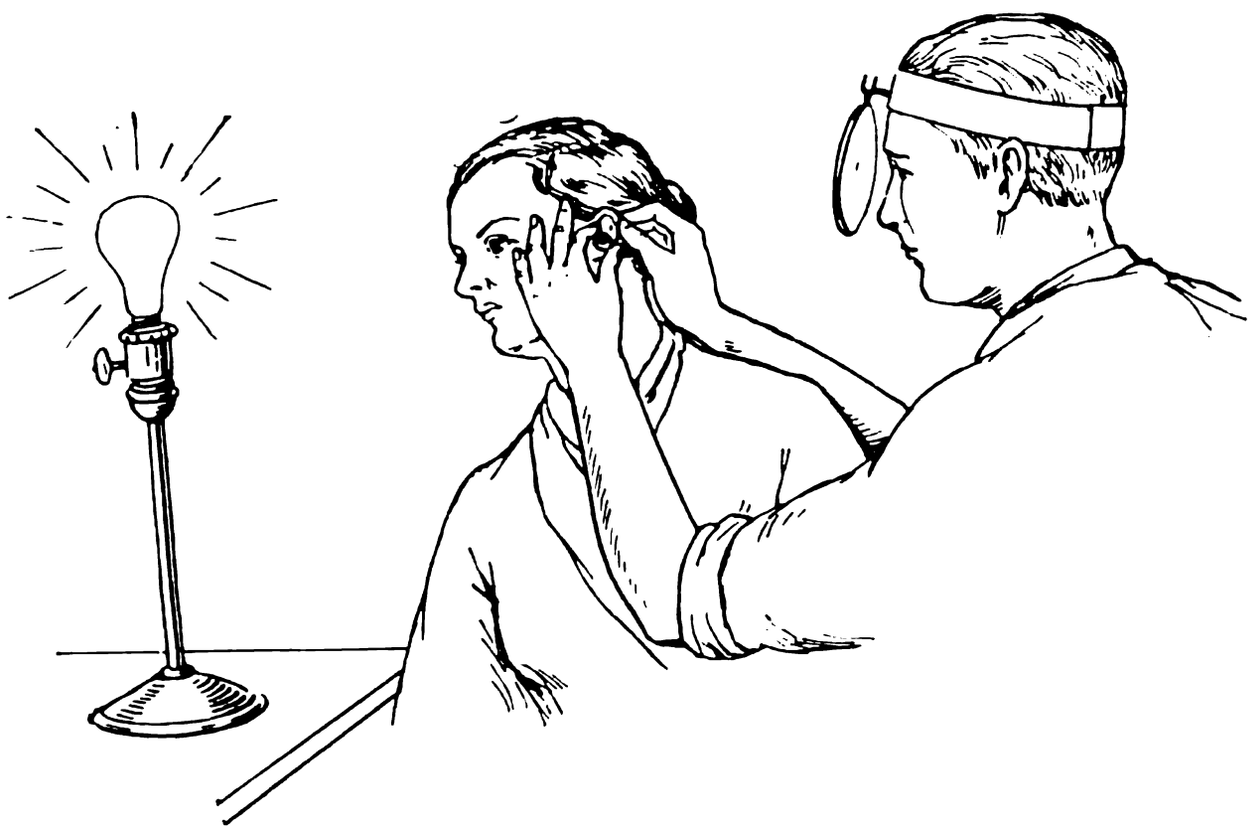


Fig. 10. Examination of Drum

Both eyes are used when making an otoscopic examination, the left eye necessarily peering through the mirror hole. Before inserting the ear speculum, one should examine the meatal opening and measure its diameter. Little children are examined with the aid of an assistant who keeps the child's head firmly pressed to his breast with one hand, and holds its arms with the other. The legs of the child are pressed between the legs of the assistant.

The ear speculum with its dilated part held between the thumb and the forefinger is carefully introduced into the meatal opening with a gentle rotation to a depth of 1 to 1.25 cm without touching the bony part, in so far as is practicable. At the same time, the pinna is pulled upward and backwards in adults and downwards and backwards in small children to straighten out the angle of the meatus.

Where there are swellings, fissures or eczema, the speculum should be introduced with particular caution to avoid pain. Lightly moving or tilting the inner part of the speculum, the examiner inspects in turn the internal parts of the auditory meatus and the entire surface of the drum. In examining the meatal canal major attention should be given to the posterosuperior wall to detect possible thickenings and even considerable overhangings extending downwards and outwards.

It is possible to examine the ear without the aid of artificial lighting only in cases of a sufficiently wide and fairly straight auditory meatus. To avoid obstructing the light with his own head, the doctor should be at some distance from the patient, although ear details will not be seen so clearly.

The normal drum is an oval disc of a pearly grey colour on which the following landmarks are clearly visible (Fig. 3, coloured Table I, Fig. 1). A whitish-yellow knob, the size of a pin-head, projecting from the anterosuperior part of the drum is the *short process* of the malleus. Two greyish-white streaks extending backwards and forward from it are the *anterior* and *posterior folds* separating the anterosuperior part of the drum, the pars flaccida of Shrapnell, from the lower tense part, the pars tensa. From the short process the handle of the malleus can be seen extending downwards and backwards. Its broader lower end terminates at the centre of the drum, the *umbo*. At otoscopy, the light rays from the mirror form on the drum a brilliant cone-shaped light reflex with the top pointed to the centre (umbo) and the base facing the anteroinferior edge of the tympanic membrane.

For convenience, in describing changes on the tympanic membrane, the latter is divided into four quadrants (Fig. 3) by an imaginary line extending the malleus handle to the edge of the drum and by an intersecting line drawn at right angles to the first through the centre of the umbo; thus forming the anterosuperior, anteroinferior, posterosuperior and posteroinferior quadrants. The drum is very closely connected with the tympanic cavity; therefore, it will reflect any existing morbid condition of the middle ear. Thus, a change in colour, for instance, redness of the drum, indi-

cates otitis media. Changes in location of the landmarks, the light cone in particular, manifest themselves in cases of drum retraction resulting from chronic diseases of the middle ear.

The mobility of the drum may be tested by the use of a pneumatic speculum (Fig. 11). The dilated end of this specu-

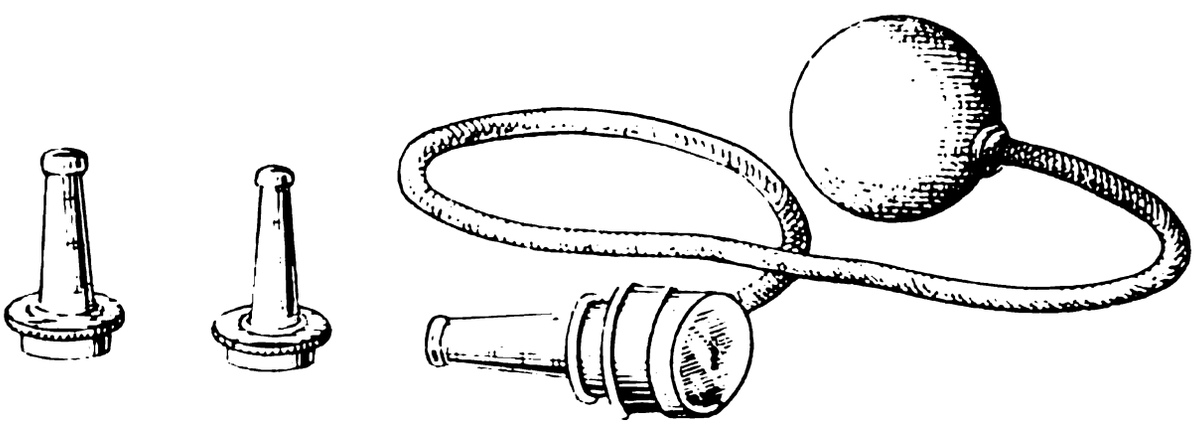


Fig. 11. Pneumatic Speculum

lum is hermetically sealed with a lens, and a tube branching from its side is attached to a rubber bulb. To facilitate observance of drum mobility through the lens, the speculum is tightly fitted into the auditory meatus, the air being alternately compressed and rarefied with the rubber bulb, causing the drum to move in and out.

At otoscopy the examination begins with the healthy ear in order to compare it with its diseased counterpart.

Examination of the Auditory Function

The functional examination of hearing is made by means of whisper and conversation voice, tuning forks and audiometers. The whisper test should be carried out in a quiet room of adequate size. Each ear is examined separately, while the other ear is closed with a finger tip. The patient turns the ear to be tested to the examiner so that he does not see the latter's face and cannot guess his words by watching the lip movements. The whisper should always be of the same intensity obtained by breathing out effortlessly and using the residual air to call out words.

The normal ear can hear whispered speech with a prevalence of low tones, i.e., consonants, labial and palatal sounds, b, p, t, m, n, at a distance of 5 to 10 m; whispered speech with a prevalence of high tones, i.e., sibilants, s, z, ch, sh, shch, is heard at a distance of up to 20 m. Estimation of hearing by the whisper test may be made by calling out numbers from one to a hundred and selecting numbers with low sounds, such as two, five, nine, and with high sounds, such as six, seven, sixty-six, etc. As the figures uttered can be easily guessed by some patients, the hearing test should rather be made with specially selected words of high and low tones. A table of such words has been compiled by V. I. Voyachek. The first group of words primarily contains low sounds heard at a distance of 5 m on the average; the second group is mainly composed of high sounds heard at a distance of 20 m. The following is a conjectural selection of English words based on the same principle.

The average furthest distance for perception of whispered speech in normal hearing is 5 m		The average furthest distance for perception of whispered speech in normal hearing is 20 m			
fun	miller	I	sigh	satchel	zealous
vow	mirror	eight	see-saw	issuant	chit-chat
brew	model	oil	snatch	cyst	child
prune	marrow	shirt	chatter	sat	chaste
more	morose	church	chassis	sharp-sighted	chastise
moon	moot	say	cheating	sacred	chest
no	number	chess	Dutchman	stitching	cheesy
noon	narrow	China	shears	signal	charter
own	narrate	Jane	chart	sittings	shellac
room	abode	jazz	syringe	stayer	sheet
Rome	adore	chirp	spike	smith	shellfish
loom	ago	cheese	side-car	Chinese	dresser
borrow	applaud	chisel	side-dish	sheath	teaching
rowing	rebuke	saint	sackcloth	siesta	giddy
run over	run out	sachet	ashes	setting	yachtsman
aroma	hurricane	itching	cherry	sad	jest
dowry	morrow	sachem	saucy	set	

By using this table one may roughly determine the nature of the ear disease. A poor perception of words of the first

group (low tones) will indicate a defect in the sound-conducting apparatus, i.e., conduction deafness. Impaired hearing of words from the second group (high tones) will point to a lesion in the sound-perceiving apparatus, i.e., nerve deafness. If whispered speech is heard at a distance of 6 to 8 m, the hearing is practically normal. If the patient does not hear the whispered speech at all, conversation voice or forced voice tests should be used. In examining unilateral deafness it is not enough to close the normal ear with a finger tip to shut out all sound. Therefore, a special noise-box (Fig. 12) is put into the normal ear to exclude its hearing altogether.

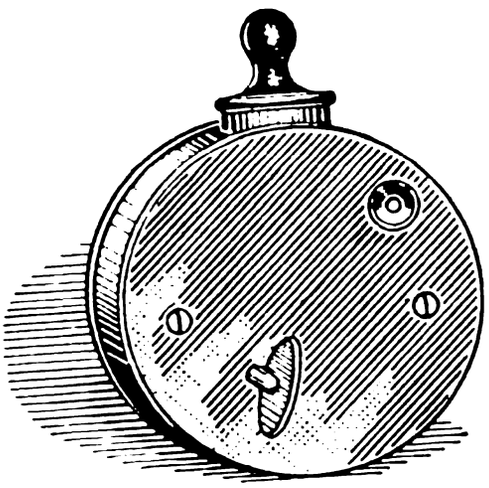


Fig. 12. Noise-box

Other methods of excluding the normal ear, such as rubbing the pinna with the flat of the open hand or shaking the finger plugging the auditory canal are less reliable.

Tuning fork tests. Hearing acuity is determined by the whisper test with comparative ease and quickness. But the differential diagnosis between the conduction and perception types of deafness, as well as a more accurate determination of hearing acuity is made by using tuning forks (Fig. 13). The tuning fork produces a clear tone without overtones. The human ear can hear in a sound range of 16 to 20,000 cycles per sec. The highest and lowest tones perceptible to the human ear indicate the higher and lower limits of hearing respectively. The tones heard between the upper and lower limits of hearing make up the so-called audible range or register. This range decreases noticeably with age, mostly due to reduction of the upper limit of hearing. A set of tuning forks can be used to examine hearing acuity for different tones, from 64 to 4,096 cycles per sec, as well as to determine air and bone conduction. To estimate air conduction, a sounding tuning fork is held near the meatal opening. When bone conduction is being determined, a sounding tuning fork is placed on the head or the mastoid process. In normal hearing air conduction is better than bone con-

duction. In medical practice, bone and air conduction is examined only with two tuning forks, of the types C_{128} and $C_{2,048}$. The following experiments can help in a differential diagnosis between the conduction and perception types of deafness.

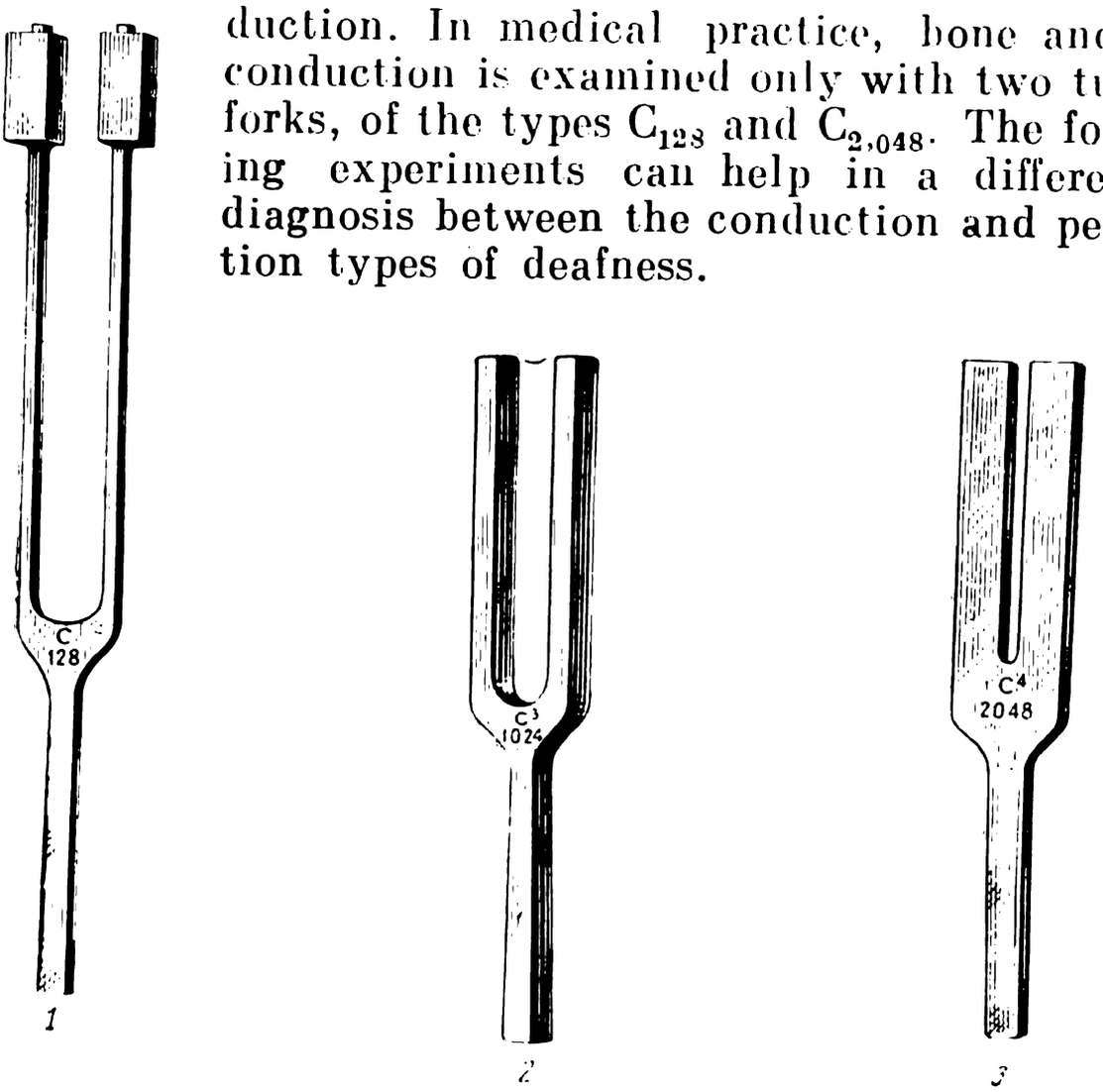


Fig. 13. Tuning Forks of the Types:
(1) 128 cycles per sec. (2) 1024 cycles per sec. (3) 2048 cycles per sec

The Weber test of sound lateralisation (Fig. 14, a). A C_{128} tuning fork is sounded and placed on the vertex, and the patient is asked to say in which ear the sound is heard best. In case of conduction deafness, such as caused by earwax impaction in the auditory meatus or otitis media, the sound will be best heard in the diseased ear. In the event of nerve deafness, the tuning fork will be best heard in the normal ear.

The Rinné test or comparison between bone and air conduction (Fig. 14, b). When a sounding C_{128} tuning fork applied to the mastoid process can no longer be heard, it is held to the ear; it then appears that the sound is heard longer by air conduction than by bone conduction, normally for 85 to 90 sec compared to 45 sec in the case of bone conduction. In such circumstances, the Rinné test is considered positive

(Rinné+); a positive Rinné is observed in patients with normal hearing, as well as in those with nerve deafness. In conduction deafness the duration of bone conduction may be equal to that of air conduction or even exceed it considerably, in which case the Rinné test is considered negative (Rinné—).

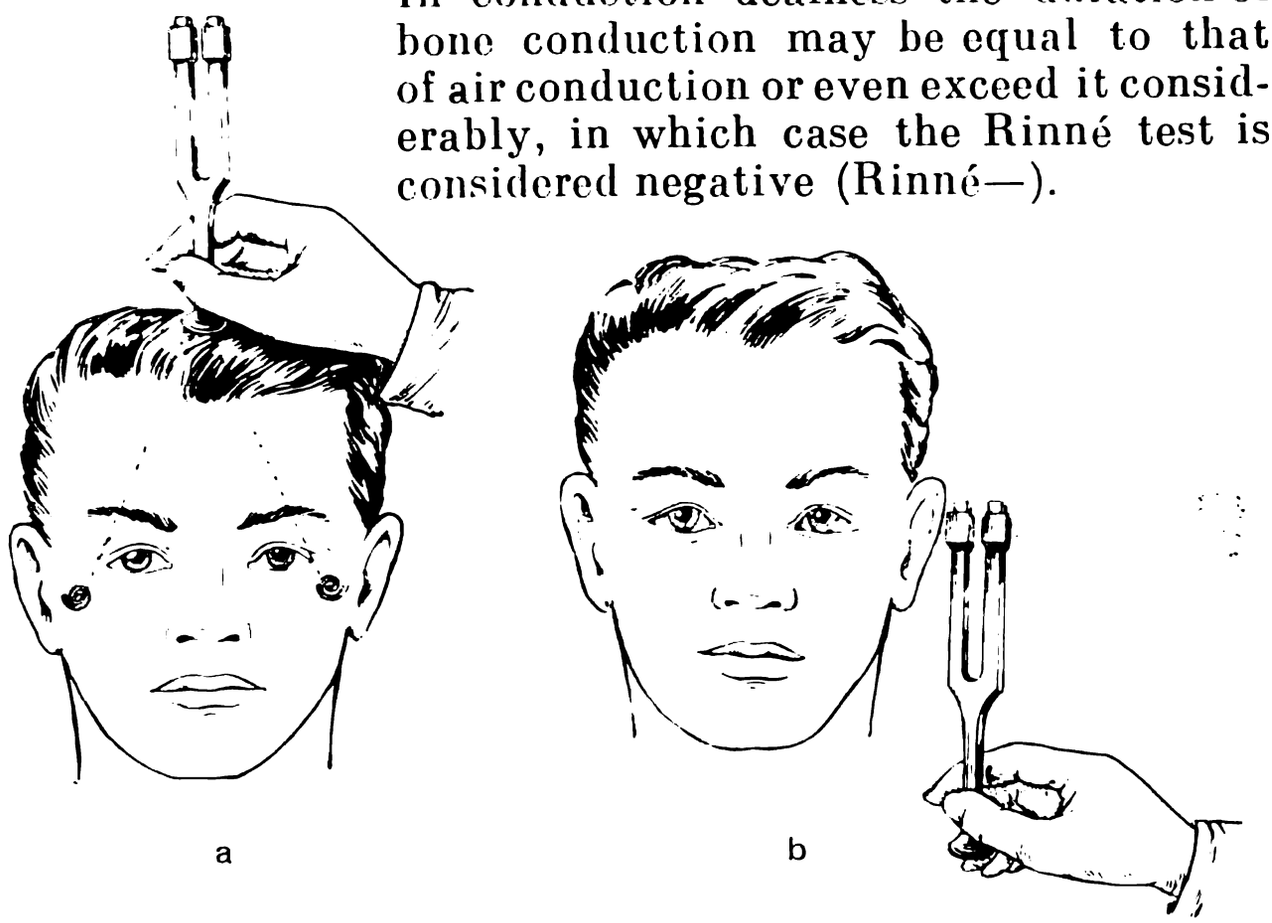


Fig. 14. Examination of Hearing with Tuning Forks:
(a) the Weber test; (b) the Rinné test

The Schwabach test compares the duration of bone conduction from the vertex or the mastoid process of the patient with the normal bone conduction of the examiner. In conduction deafness, bone conduction is lengthened, whereas in nerve deafness it is shortened.

The acuity of hearing for high tones is determined with a C^4 tuning fork of 2,048 cycles per sec. A certain high tone loss is noticeable in deafness due to old age and in nerve deafness.

A more accurate and quicker examination of hearing is made with a special instrument called the *audiometer* which can be used to estimate hearing acuity within the entire tone range important in the estimation of hearing.

Examination of the Vestibular Function

Examination of the vestibular function is preceded by questioning and examining the patient. Patients with vestibular disturbances usually complain of giddiness, unsteady gait, as well as occasional sickness and vomiting. Examination of the patient may reveal a nystagmus often associated with a functional vestibular disturbance. Vestibular nystagmus may be observed in a patient looking sideways and sometimes in a patient staring straight ahead. Disorders in co-ordination, static equilibrium and balance of walking are also likely to happen. Special clinical tests are used to examine the vestibular function. These tests are based on procedures for artificial stimulation of the vestibular receptors to produce nystagmus. There are three basic tests of this kind, the rotation, caloric and compression tests.

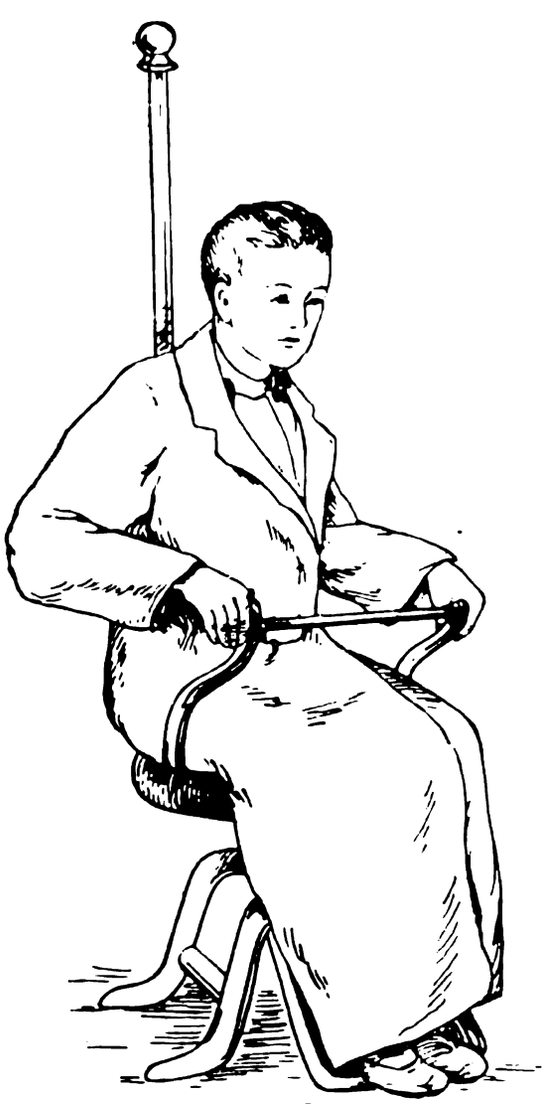


Fig. 15. Rotating Chair

The *rotation test* involves turning the patient's body around the vertical axis to cause endolymph movements in the semicircular canals, which stimulate the receptors and produce nystagmus. The patient is seated in a chair (Fig. 15) which can be rotated horizontally and is revolved at a speed of 10 revolutions in 20 sec. This stirs up the endolymph in the semicircular canals. When the chair is stopped abruptly after rotation to the right, the patient will have leftward nystagmus; if the procedure is reversed the nystagmus will be in the opposite direction. So that the nystagmus may be studied, the patient is directed to look at the examiner's finger, which is held at a distance of 30 cm towards the side where the nystagmus is expected.

In most people with normal vestibular sensitivity the duration of post-rotational nystagmus is, on the average, 30 to 35 sec. Among the disadvantages of this test is that rotation stimulates both labyrinths, though with unequal force.

The *caloric test* is based on the phenomenon of endolymph movement in the semicircular canals under the effect of cooling or heating by artificial means.

During this test each labyrinth is examined separately. Each ear in turn is syringed with water; a cold douche at 16° to 30°C causes nystagmus to the side away from the ear being tested, whereas a warm douche at 38° to 41° C causes nystagmus in the opposite direction.

Absence of nystagmus in the caloric test may indicate a loss of vestibular sensitivity.

In case of dry perforation one should abstain from the caloric test for fear of provoking a relapse of suppurative otitis.

Some pathological conditions of the labyrinth are likely to produce a nystagmus caused by air compression or rarefaction in the auditory canal. This is known as the *compression test*. If there is a fistula or a bone defect in a labyrinth wall (external semicircular canal), the air compressed with a bulb in the auditory meatus causes nystagmus towards the diseased ear, whereas aspiration produces nystagmus to the opposite side. This phenomenon is called the fistular symptom.

GENERAL METHODS OF NURSING AND TREATMENT OF EAR DISEASES

Diseases of the ear, like those of the nose, pharynx and larynx should not be treated without regard to the general condition of the body.

Therefore, their treatment should be combined with other methods to improve the patient's general state of health.

Among these are prescription of analgesic and sedative drugs with a favourable effect on the central nervous system, sulfonamides and antibiotics, as well as the use of other means to raise general body resistance.

Frequently, the treatment prescribed by a doctor is unsuccessful primarily because it has been wrongly applied.

To a large extent the success of medical treatment is dependent on the correct and skilful fulfilment of the doctor's instructions by the junior medical staff. The simplest methods of nursing and treatment in ear diseases usually do not require special contrivances and intricate instrumentation, and can easily be applied in hospitals and out-patient clinics. These simple procedures refer to cleansing of the ear, introduction of medications into the auditory canal, application of hot compresses, etc.

Methods of Cleansing the Ear

Before examining the drum, it is often necessary to cleanse the external auditory meatus of wax, pus or crusts formed by desquamated cells and dried pus. The cleansing of the external auditory meatus is part of medical treatment and can be performed by the wet and dry methods.

The *wet method of cleansing* or syringing the ear is applied in cases of acute or chronic suppurative otitis media where pus exudation is so abundant that its removal by drying with cotton wool cannot be complete when carried out by the patient or would necessitate too much qualified nursing care.

The ear may be washed with an ear syringe or an ordinary rubber bulb, which is less dangerous, particularly when the syringing is carried out by lay people. There are two types of rubber bulbs: all-rubber bulbs with a broad tip, difficult to insert into the auditory canal, and bulbs with a bone tip which are more suitable for this purpose. A thin 3-4 cm long rubber tube cut off obliquely at the end should be put on the bone tip to avoid accidental injury to the walls of the external auditory meatus. The ear is cleansed with warm antiseptic lotions, mostly 3% boric acid solution. The patient holds a kidney basin used in the procedure tightly pressed to the appropriate side of his neck. To straighten out the auditory canal of an adult patient to a greater extent, the pinna is pulled by the left hand backwards and upwards, while the rubber tip of the bulb is introduced into the ear by the right hand no further than 1 cm. Successive jets of warm liquid at 37 °C are directed under low pressure along the posterior wall of the auditory canal. When filling the rubber bulb

with liquid, it should be pressed to make sure that it contains no air, for otherwise air bubbles mixed with liquid will cause an annoying noise in the patient's ear. After syringing, the patient's head is inclined to the side to let the liquid pour out of the ear. The residual liquid is removed from the far end of the auditory meatus with cotton wool wrapped round a probe. Wax removal by syringing is described in the chapter "Cerumen Impaction in the Ear".

The *dry method of cleansing* the ear is more expedient than syringing, as the latter is very likely to introduce a secondary infection into the ear. The auditory canal is dried or mopped with cotton wool to remove pus in cases where pus discharge is small or syringing is contraindicated in consequence of skin irritation in the meatal walls, for instance, in eczema or boils. To prevent recurrence of the disease, the ear is mopped dry with cotton wool, soaked in 70% alcohol. Slender probes with threaded tips are used to wipe and dry the inside of the ear. Smooth and eyed probes are not suitable for this purpose. The cotton wool should be tightly wrapped on the probe so as to cover its sharp tip to prevent injuries to the meatal walls or the tympanic membrane.

Before cleansing the ear, the hands should be washed with soap; the cotton wool should be hygroscopic and sterile. The probe with a cotton wrapping should be sterilised in the flame of an alcohol lamp.

The ear is wiped in the following manner. The left hand is used to hold the ear speculum and pull the pinna backwards and upwards, or backwards and downwards when dealing with small children, and the probe tip wrapped in cotton wool is carefully introduced by the right hand into the auditory canal, which is then cleansed up to the drum with light probe rotation. This rotatory motion of the probe helps the cotton wool absorb secretion. This procedure is repeated until the cotton wool taken out of the ear is fully dry; only then may drops be instilled, if necessary.

The dry method of treatment of suppurative otitis media with the aid of ear tamponade is intended to drain the middle ear of harmful matter and in certain cases is one of the best ways to cure suppurative otitis. It can also be used after

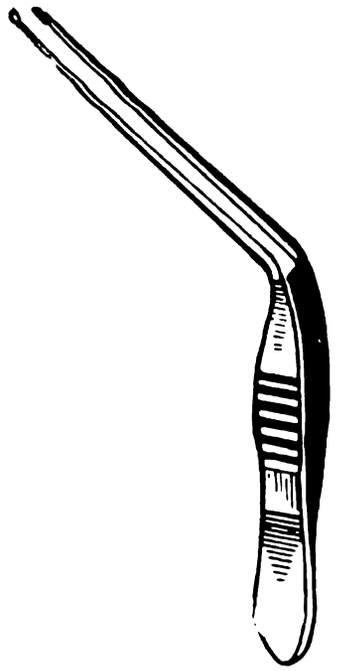


Fig. 16. Angular Forceps

syringing or drop instillation, but in such cases the auditory canal should be dried before the introduction of a tampon. The ear tampon is a folded narrow gauze strip, not more than 5 to 7 cm in length. Ear tamponade is performed in the following manner. The pinna is drawn backwards and upwards by the left hand to straighten and dilate the auditory canal. The ear tampon is gripped with an angular forceps (Fig. 16), at a distance from its front margin, and is carefully pushed forward along the auditory meatus. Upon removal of the forceps, the gauze tampon is again gripped at 1 to 1.5 cm from its margin, and is pushed with great caution further into the meatus until it comes into contact with the drum. The ear tampons should lie loosely in the auditory canal to prevent pus congestion. Ear tampons should be changed five to six times a day where there is copious pus discharge and once or twice a day when the pus is scanty. Naturally, all the above-mentioned procedures of curing ear disease should be in strict accordance with antiseptic rules.

Medication

Medications used in ear diseases take the form of drops, ointments and powder. The most common medical procedure in ear disease is instillation of drops into the ear. If the auditory canal contains pus, drops should be instilled only after it has been thoroughly cleansed by repeated dry wiping with cotton or by syringing. Prior to instillation the drops should be heated up to body temperature to avoid stimulation of the vestibular apparatus. When drops are being instilled the patient should incline his head to the side away from the ear treated. The pinna is pulled by the left hand backwards and upwards, and 5 to 10 drops of medicine are instilled with a dropper or teaspoon into the auditory canal. Drops should be instilled two or three

times a day and allowed to remain 10 to 15 minutes at a time; the ear is then held down to let the drops out. After this the auditory canal is dried, as described above, and filled with a loose gauze tampon, if prescribed by the doctor.

Ointment is applied only in diseases of the external auditory canal and the auricle, such as dermatitis or eczema. Those most commonly used are white and yellow mercury ointments, as well as zinc and streptocide ointments which are spread over the meatal walls with cotton wool wrapped on a probe or a thin match. A tampon with ointment is sometimes left for 15 to 30 minutes in the auditory canal. The latter should not be packed full with ointment, because this will interfere with ear secretion drainage.

The *insufflation of powder drugs* is made by a doctor or may be entrusted to junior medical or nursing personnel, and in some cases to the patient's relatives. Powder insufflation should be preceded by a thorough cleansing of the auditory meatus of pus and debris by the wet or dry methods. Very fine powders of boric acid, streptocide or sulfadiazine in pure form or with additions of other drugs, such as penicillin and synthomycin are widely used for insufflation. Insufflation is performed by means of different types of insufflators (Fig. 17) or, if the latter are not available, by means of a make-shift paper funnel or an ear speculum whose end is plunged into the powder to take it up in small quantity. The end of the speculum or funnel is inserted into the ear, and the powder is blown inside with a rubber bulb. Metal tips of insufflators should be sterilised in boiling water, and rubber tips should be wiped with cloth soaked in alcohol or 4% carbolic acid solution. In the process of

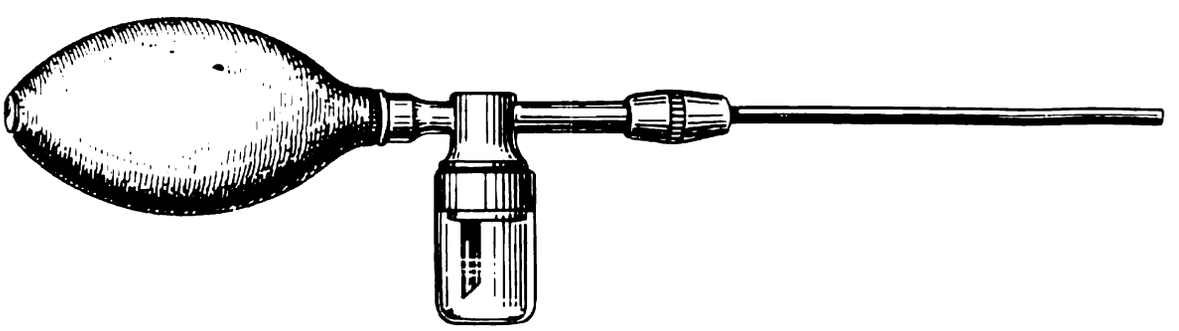


Fig. 17. Insufflator

insufflation the auditory canal should be kept straightened by pulling the pinna backwards and upwards, while the powder should be made to form a thin uniform layer without lumps that may obstruct pus drainage.

The *hot compress* consists of three layers: (1) a wet layer, that is a piece of hygroscopic fabric soaked in water or solution; (2) a layer of waterproof material, preferably oilskin, or wax-paper as a substitute; (3) a layer of fabric with poor heat conductivity, such as cotton wool, woollen fabrics or flannel. The first layer is applied to the skin and is superimposed by the second and third layers which should both be larger than the first and cover it all over. A head bandage protecting the ear is then applied. A compress thus placed reduces and even stops the emission of heat from the skin, so that water vapours of body temperature form between the top and bottom layers. This causes a uniform and protracted dilatation of vessels in the skin, as well as in deeper organs and tissues, which is followed by intensified metabolism, resolution of inflammatory infiltrate or limitation of suppuration.

DISEASES OF THE EXTERNAL EAR

Burns and Frostbite of the Auricle
(*Combustio et congelatio*)

There are three degrees of burns of the auricle: the light, severe and very severe. Severe burns may result in parts of the auricular cartilage becoming necrotic, leading to their destruction and consequent ugly ear deformities. Burns are treated by common surgical methods. Frostbites of the auricle are more frequent than burns. Light frostbites cause intense redness and slight swelling of the skin, followed by severe burning and pain, particularly after entering a warm room. More severe frostbites cause blebs on the skin, and very severe freezing produces necrosis of the skin and cartilage.

Treatment. Light frostbite is treated by gradual thawing of the ear, carefully wiping it with an alcohol-soaked cloth and painting it with some sort of sterile oil. To relieve

itching and burning the ear skin is painted with 5% iodine tincture or camphor ointment.

Rp. Camphorae tritae 0.2
Lanolini 10.0
Vaselini albi 15.0
m. f. ung.
D.S. To be applied a few times daily

The blebs should be opened and covered with a coat of zinc or streptocide ointment spread on a bandage. Surgical removal of necrotic soft tissues and cartilage should be followed by antibiotic and sulfamide treatment.

Erysipelas

Skin abrasions and other injuries of the auricle are likely spots of penetration by streptococci. Erysipelatous inflammations are especially frequent in persons with otitis media, weeping eczema, or dermatitis of the external auditory canal.

In some cases erysipelas of the auricle is the result of autoinfection from the face or the hairy part of the head.

Local symptoms, such as large swelling and erysipelatous redness, are preceded by fever, the onset of malaise and headaches. Correctly treated, the disease lasts three to four days. On the first day the local reaction of the skin on the mastoid process may be mistaken for a symptom of mastoiditis.

Treatment. Treatment is with streptocide in a dose of 3 to 4 g per 24 hours taken orally in equal portions once in every four hours; with intramuscular injections of penicillin in doses of 100,000 units 5 to 6 times in 24 hours; with levomycetin taken orally in 0.5 g doses 4 times in 24 hours, as well as with biomycin, ultraviolet light in an erythema dose and by painting the skin with neutral ointments to reduce painful tension.

Rp. Biomycini hydrochlorici 100,000 units
D. t. d. N. 20 in tabs.
S. Five doses of 2 tablets in 24 hours

Furuncle of the External Auditory Canal

(*Otitis externa circumscripta*)

Furuncle of the auditory canal is a skin inflammation, mainly confined to the cartilaginous portion, caused by inflammation of a hair follicle and a sebaceous gland.

Injuries to the skin of the auditory canal caused by nails, matches or other sharp objects facilitate the entrance of staphylococci into hair follicles. In some cases, furuncle of the external auditory meatus is a symptom of general furunculosis due to a metabolic disturbance, emaciation or malnutrition. Most boils occur on the anteroinferior wall of the auditory canal.

Symptoms. The process begins with pain of increasing intensity in the ear, which may be so severe as to deprive the patient of sleep. Chewing increases the pain which is at times referred to the teeth, forehead and back of the head. Body temperature is normal or subfebrile. The cartilaginous portion of the auditory meatus is considerably narrowed by a cone-shaped infiltrate on one of its walls. Pressure on the auditory canal, particularly on the tragus, as well as any traction on the pinna is very painful. The hearing is usually normal. When the boil is ripe, it bursts into the auditory canal, and if there is no relapse, this is followed by healing.

Treatment. In the dense infiltration stage, hot compresses are advisable to help bring the process to maturity and sometimes to abortion. A slender gauze strip soaked in 10% Burow's solution or pure alcohol is packed into the auditory canal. These ear tampons should be changed every two or three hours.

In addition to disinfection of the auditory canal, this method of treatment makes the boil ripen more rapidly. In addition a hot compress or heat from the "sollux" lamp also serves to relieve the pain; ultraviolet light in a suberythema dose is also applied once or twice. Only large abscesses causing massive edema and severe pain may be incised. In rare cases the process may spread beyond the auditory meatus and form abscesses behind the ear, or it may extend through Santorini's fissures to the parotid area. Treatment of these conditions, apart from surgical

incision, is with penicillin and streptocide to be taken in 0.3 g doses 8 times in 24 hours.

To prevent relapses, the meatal skin is wiped with cotton wool soaked in 70% alcohol or painted with 2% yellow mercury ointment. This is combined with general body invigoration by means of arsenic, brewer's yeast and auto-hemotherapy.

Otitis Externa Diffusa

Otitis externa diffusa rarely occurs as a primary disease. It usually follows acute or chronic suppurative otitis media in consequence of inadequate or incorrect treatment.

Skin inflammation in the auditory canal causes it to swell and narrow down concentrically. The canal fills with desquamated epidermal cells and purulent exudate. In the acute stage there is severe pain and tenderness of the ear, in the chronic condition pain may be completely absent. The hearing is impaired only in case of otitis media.

Treatment. In the acute stage, the ear should be syringed with a warm boric acid solution and dried with cotton wool wrapped on a probe, as well as with gauze tampons. On the first and second days, the ear is packed with tampons soaked in 10% Burow's solution and replaced every one or two hours; later, dry tampons can be used. A dry tampon should be removed from the canal before it gets fully saturated with exudate. In chronic inflammations with superficial skin ulceration, it is advisable to apply 3-5% silver nitrate solution, sulfonamide insufflation or ear tampons soaked in sulfonamide emulsion. In concurrent suppurative otitis media, the pus in the canal should be dried up by careful and frequent application of narrow gauze tampons.

In otitis externa attended with itching it is important to correct the patient's diet by forbidding highly-spiced, canned or smoked foods and prescribing a diet with a low salt content, nicotinic acid and vitamins C and B_2.

Rp. Ac. nicotinici 0.05
Sacch. lactici 0.2
D. t. d. N. 12
D.S. One powder dose, three to four times daily after meals

Itching often ceases after administration of antihistaminic drugs, such as dimedrol taken in three doses of 0.025 to 0.05 g daily for 7 to 10 days.

Eczema of the External Ear

(*Eczema auris externae*)

Eczema may be due to various local causes, such as skin irritation in pus discharge from the ear, mechanical, thermic and chemical irritations, as well as to general diseases associated with dysfunction of the endocrine glands, metabolic disturbances, as exudative diathesis, gout, etc. The acute forms of eczema are accompanied by itching, inflammatory swelling, erythema and formation of blebs. These burst and form weeping lesions and fissures covered with crusts.

Eczema is prone to spread and become chronic, in which case it is accompanied by skin infiltration, ulceration and often causes the auricle to swell and the auditory canal to become narrow sometimes. After recovery eczema may well recur due to quite insignificant causes. To prevent any such relapse the patient is forbidden to scratch the ear or to wash it with soap and water, or to wipe it with cotton wool or other materials.

Treatment. The primary aim of local treatment is to relieve itching, for which purpose styptic lotions are prescribed to be packed into the ear at regular intervals with tampons soaked in 5% Burow's solution or lead water; other remedies are Lassar's paste and the dust *Acidi salicylici* 0.5, *Amyli tritici*, *Zinci oxydati* $\overline{aa}$ 10.0. Two per cent salicylic ointment is used to soften and remove the crusts.

Weeping eczema is treated with 2% silver nitrate solution or a 1% solution of brilliant green with subsequent application of the same ointments. Also effective are 2% boric tar and 2% boric naphthalan pastes. If possible, the ear should not be bandaged, as this hampers recovery. Ultraviolet light is helpful in some cases.

Apart from local treatment, general treatment is also important, particularly for children in whom eczema is often caused by an alimentary disturbance.

Cerumen Impaction in the Ear

In certain conditions wax accumulation in the ear may obstruct the auditory canal. Normally, cerumen secreted by numerous ceruminous glands dries into crumbs and falls out.

The causes of abnormal impaction of earwax may be (a) excessive glandular secretion, as a result of local skin irritation brought about by matches, the tip of a folded towel used to remove wax, or excessive wax secretion normally present in the patient; (b) narrowness of the external auditory canal, which prevents normal wax expulsion; (c) abnormal properties of earwax, such as its excessive viscosity and tendency to stick to the meatal walls.

Earwax impaction may be extensive and at the same time may have no ill effect. If a cleft, however narrow, remains between the meatal wall and the cerumen mass, the hearing will be normal. But should a small amount of water get into the ear in this condition, as occurs in washing the head or in hot bathing, the hearing will sharply deteriorate, for the water will cause the wax to swell and obstruct the passage completely. Patients also complain of ear noise or tinnitus, and distorted hearing of their own voice, which resonates in the stopped-up ear. There may often be reflex cough from vagus nerve stimulation.

Diagnosis. Cerumen impaction can be easily detected by otoscopy, which reveals the mass of red-brown or dark-brown wax occluding the passage. A probe is used to determine whether the wax is hard or soft.

Treatment. The wax is removed from the ear by syringing it with warm water. Pending this operation the ear should be investigated for the presence of pus discharge since in this case a dry perforation of the drum is likely to remain. Should this be so, syringing may aggravate the process and cause pus discharge to recur. In such cases it is preferable to remove the wax with a cerumen hook or a ring curette.

Syringing should be performed with water at 37°C to avoid stimulating the vestibular apparatus, which will give rise to discomforting sensations in the patient, such as vertigo, nausea, vomiting, etc. The metal-piston syringe

(Fig. 18) should have a capacity of 100 ml. The stream of water should be directed at intervals along the posterior wall of the auditory canal, with the pinna drawn backwards and upwards (Fig. 19).

To prevent the syringe tip causing acci-

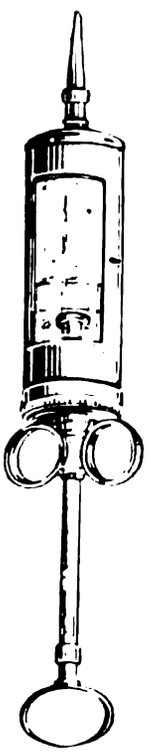


Fig. 18. Ear Metal-Piston Syringe

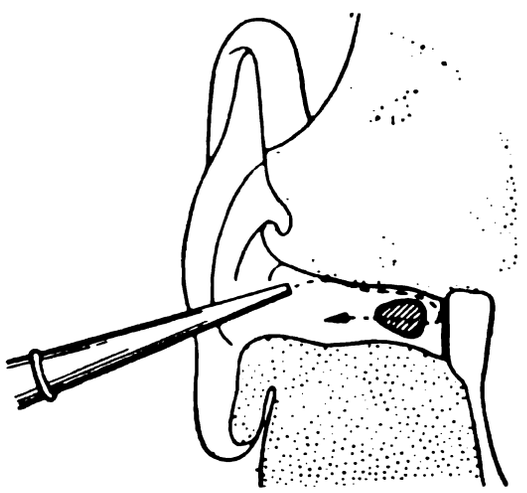


Fig. 19. Direction of Stream in Syringing

dental injury to the meatal wall or even the drum, the left hand pulling at the pinna should at the same time form a finger support for the syringe to avoid a sudden jerk of its tip into the meatal opening. A stream of adequate pressure will wash out the wax in a single lump or in small crumbs. The meatus should then be dried with cotton wool wrapped on a probe.

If repeated syringing fails to remove the wax, it should be softened by instillation into the ear, for two to three days, of alkaline drops heated to about 37 °C and allowed to remain in the ear for 10 to 15 min.

Rp. Natrii bicarbonici 0.5
Glycerini
Aq. destill. aa 5.0
MDS. A warm dose of ten drops to be taken three times daily

The patient should be warned that following drop instillation, the stopped-up feeling in the ear may increase as a result of wax swelling.

Skin diseases of the external auditory canal often cause extensive desquamation of the epidermis, which is accom-

panied by the so-called epidermoid impaction of hard consistency closely adherent to the meatal wall. This impaction can be picked out with a blunt hook, a procedure requiring experience, or washed out by syringing after softening it with salicylic alcohol drops (*Acidi salicylici* 0.1, *Spiritus vini rectificati* 10.0).

Prophylaxis. To prevent wax impaction in the ear, the patient should be warned against removing earwax with hairpins, a rolled tip of a towel, etc., as the latter serve to push the wax deeper into the auditory canal. Moreover, attempts to pick out wax with the aid of sharp objects may lead to an injury of the meatal wall and drum.

Foreign Bodies in the Ear

Foreign bodies in the ear are mostly confined to children of three to seven years of age.

Children are liable to insert into their ears various small objects, such as peas, buttons, beads, sunflower seeds, fruit stones, etc.

In adults, the most common foreign bodies are fragments of matches, grains of cereals, bits of cotton, wood, metal, coal, etc.

Occasionally, a small insect, such as a bug or a cockroach, may crawl into the ear.

Symptoms. Smooth objects which do not injure the meatal wall may remain in the canal for a long time without causing symptoms.

Objects with sharp, jagged edges, as well as live creatures whose wriggling in the canal is most irritating, may cause pain and noise in the ear.

The foreign body itself, however, is not so dangerous as an unskilful attempt to extract it.

Treatment. Removal of the foreign body must, of course, be preceded by

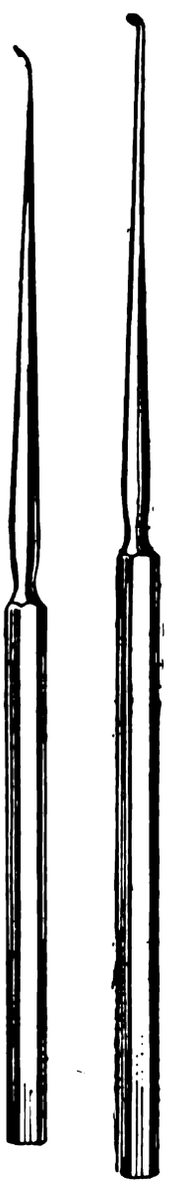


Fig. 20. Sharp and Blunt Hooks for Removal of Foreign Bodies from the Ear

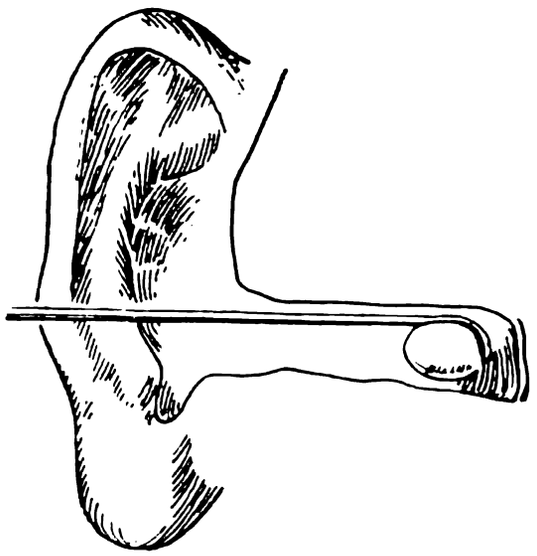


Fig. 21. Removal of Foreign Body from the Ear

inspection to determine its nature. Syringing, as in wax impaction, is the most reliable procedure for removal of a foreign body from the ear. The stream of water should be directed into the slit between the meatal wall and the foreign body. If syringing fails, the foreign body should be extracted with a small blunt hook (Fig. 20). This method requires great care and firm fixation of the patient's head; therefore, a short general anesthetic should be applied to children. The hook should be well-lit and always visible when being inserted flatwise between the meatal wall and the foreign body. Only after the hook has reached behind the object, can it be turned towards the middle, as shown in Fig. 21, and pulled back to remove it. This operation should be carried out by a skilled physician. Swollen and firmly-wedged foreign bodies, such as peas, beans, etc., are preliminarily shrunken by repeated instillation of pure alcohol into the ear.

Pincers or forceps should never be used to extract objects of round shape, as this will only result in the object being pushed deeper into the canal.

Removal of foreign bodies with instruments should only be carried out by skilled specialists, as unskilful handling of the instruments can lead to grave complications.

All the above-mentioned methods of removal of foreign bodies apply where no complications exist, where there is no considerable irritation and swelling in the meatal wall due to a foreign body or, more frequently, due to an injury resulting from an abortive attempt at its removal. If the patient's condition permits, the removal should be postponed for a few days until the inflammatory symptoms in the auditory canal subside due to drop instillation (10% Burow's solution or alcohol drops in the event of swollen foreign bodies).

Should failure to extract the object be followed by an

acute inflammation and swelling of the meatal wall or perforation of the drum, with the object deeply wedged in the bony portion of the canal, in exceptional cases a surgical operation will be required to extract the foreign body. This operation is particularly advisable if the foreign body has caused pus congestion giving rise to high temperature, severe headaches, vomiting, giddiness, etc., and if there is a danger of intracranial complication, above all, of purulent meningitis. The operation is performed under penicillin cover. After a 3 to 4 cm long incision has been made behind the pinna and carried down to the bone, the auditory canal is detached and incised along a grooved probe. Direct inspection and removal of the foreign body is thereby made possible.

Live insects are first drowned by filling the canal with chloroform water or a few drops of some oil, then washed out or removed with forceps.

INFLAMMATIONS OF THE MIDDLE EAR

Acute Catarrh of the Eustachian Tube

(*Catarrhus acutus tubae auditivae*)

Inflammations of the nasal and nasopharyngeal mucosa, as in acute coryza, influenza and other diseases, are very likely to extend to the mucous membrane of the Eustachian tube which, together with the middle ear cavity, forms a kind of nasopharyngeal diverticulum.

An inflammatory swelling of the tubal walls causes obstruction of air passage to the tympanic cavity in swallowing. Tubal obstruction also occurs in edematous hypertrophies of the posterior ends of the inferior conchae, nasopharyngeal tumours; tubal obstruction is particularly frequent in children with adenoid hyperplasias, as well as in a number of other diseases.

The *symptoms* of obstruction of the Eustachian tube detected by otoscopy may result from changes in the tympanic cavity. Cessation of air supply or inadequate ventilation will result in the tympanic cavity air being partially absorbed by the mucosa at the expense of oxygen, which is

followed by an air pressure loss in the middle ear. The disturbance of pressure balance on both sides of the drum will cause the latter's retraction.

The subjective symptoms are loss of hearing acuity, a feeling of fullness in the ear and a crackling sound heard when swallowing; the patient may sometimes imagine he hears the echo of his own voice; this is known as autophony. Where there is transudate in the middle ear, the patient will complain of a sensation of fluid in the ear. Body temperature is usually normal, ear pain is slight or completely absent.

Otoscopical examination of drum retraction reveals that the handle of the malleus takes a more horizontal position and looks shorter in perspective, the short process sticks out sharply, the anterior and posterior folds leading from it have a distinct outline. The light cone changes in form and becomes shorter to appear as a dot or disappear altogether. An acute obstruction of the Eustachian tube is often followed by hyperemia of the mucous membrane ex vacuo and appearance in the tympanic cavity of transudate whose level may sometimes be observed in otoscopy.

Treatment. This consists in the removal of the basic cause of tubal obstruction. Tumours, hypertrophies and adenoid hyperplasias in the nasopharynx are removed by surgery. Acute inflammations of the nasal and nasopharyngeal mucosa are treated with various vasoconstrictive and anti-inflammatory remedies.

Cocaine-ephedrine drops are prescribed for instillation into the nose.

Rp. Cocaini muriatici
Ephedrini hydrochlorici $\overline{aa}$ 0.2
Sol. Acidi borici 3% 10.0
MDS. One dose of six drops three to four times daily in each nostril

Penicillin ointment can also be used, as well as oral aspirin.

Simultaneously local heat treatment is given through the application of hot compresses to the ear and its irradiation with a "sollux" lamp. This treatment restores nasal respiration, serves to reduce swelling in the tubal mucosa; hence the transudation in the middle ear resolves. Restora-

tion of the tubal function and normal pressure in the tympanic cavity is helped by inflations which are best used as soon as the acute inflammation in the nose has subsided.

Acute Otitis Media

(*Otitis media acuta*)

Acute otitis media is a most common disease, particularly in children. According to their clinical course, acute cases of otitis media are divided into simple and perforating.

Both forms of acute otitis media represent two stages of a single process. This division, however, has proved valid in practice, as it gives an idea of the gravity of the inflammatory process and of its course. Moreover, this division, as will be shown later, is also justified in relation to treatment, which will differ according to each form. Simple otitis, though producing exudation in the middle ear, will not cause drum perforation and pus discharge, as is the case in perforating otitis. Therefore, the first form of otitis is often, though with insufficient reason, called catarrhal, whereas the second form is known as suppurative.

Etiology. Acute otitis media is caused by pathogenic bacteria entering into the tympanic cavity. Various microorganisms, pathogenic included, may always exist even in a healthy nasopharynx. General causes of a reduced body resistance and local pathological changes may both be among the factors favourable to the infection penetrating and developing in the middle ear cavity.

Among the general causes is the common cold which changes the functional condition of the whole body, as well as acute infectious diseases, such as influenza, measles, scarlet fever, etc., which reduce body resistance. Other causes may refer to acute inflammatory processes with local symptoms, such as acute catarrh of the upper respiratory tract, acute rhinitis, acute catarrh of the nasopharynx, tonsillitis, etc. The whole variety of pathological changes in the nose and nasopharynx, such as hypertrophy of the posterior ends of the inferior conchae, diseases of the paranasal sinuses, ozena, nasal polyps, adenoids, chronic tonsillitis, etc., may contribute to the development of acute otitis. Sometimes, acute otitis follows a tamponade of

the nasal cavity, its far region in particular, as well as an unskilful nasal douche or surgical interference and cauterisation in the nose.

The infectious germs spread up the Eustachian tube and into the middle ear in coughing, sneezing and primarily in an energetic blowing of the nose. This route of infection, known as the tubal route, is the most common. Much less frequently the germs enter through the blood stream, which occurs in infectious diseases. Cases of their entrance through an injured drum are exceedingly rare.

The pathogenic agents of acute otitis media are, in order of importance, all types of streptococci, staphylococci and pneumococci. The latter are confined to children.

In light cases, pathological changes in the middle ear are limited to marked congestion of the mucous membrane, light swelling and edema of the latter together with, at first, serous and then purulent exudation. More severe forms of acute otitis produce marked swelling of the mucosa, so that it may be thickened 15 to 20 times above normal and in some cases cause complete obstruction of the middle ear cavity; microscopical examination will reveal dilatation of small vessels, the capillaries of the subepithelial layer, and profuse infiltration of all mucous layers with round cells. The inflammatory exudate quickly becomes purulent.

From the very outset the drum is involved in the process, it becomes swollen, congested and loses its outline.

Symptoms. The symptoms of acute otitis media vary considerably, both in severity and in the rate of aggravation.

The patient feels severe pain which radiates to the vertex and occiput. The pain may be mild at first and increase gradually, or it may become intense at once. The fullness or stopped-up feeling in the ear which develops at first quickly turns into deafness of varying degrees accompanied by tinnitus, which later on is often felt as a most oppressive throbbing.

The physical signs revealed by otoscopy are of substantial importance in the diagnosis of acute otitis media. All the stages of inflammatory process in the tympanic cavity are reflected on the drum as if it were a mirror. Inflammatory changes begin with a slow-growing congestion of the drum. The first sign is a dilatation of vessels along the handle

of the malleus followed by a radial injection of the vessels from the drum margins towards the centre (see coloured Table I, Fig. 2) and then the entire drum membrane becomes congested and bright-red all over its area. The drum becomes thinner, loses its contours and begins to bulge into the auditory meatus (coloured Table I, Fig. 3). An increase of exudate in the tympanic cavity causes the drum to protrude still more, particularly in its posterior aspect. The tympanic membrane gradually grows thinner and turns yellow at its most prominent point, reflecting the purulent exudate. If an incision (paracentesis, myringotomy) has not been made in such cases, a spontaneous perforation of the drum will follow with discharge of the exudate. Later the epithelial layer of the drum macerates and separates, as a result of which the drum may assume a greyish colour which serves as a background for the congested portions of the still inflamed drum to be seen through cracks in the epidermis. The moment of drum perforation by the accumulated exudate is of decisive importance, as it serves to differentiate between simple (catarrhal) and perforating (suppurative) forms of acute otitis media. While the drum is still intact even though purulent exudate is present in the middle ear cavity, the inflammation is provisionally termed simple or catarrhal.

Following paracentesis or a spontaneous perforation of the drum, the auditory canal fills with discharge which at first is serosanguineous and then becomes mucopurulent to turn finally into frank pus secreted in an ever-decreasing quantity.

The aggravating inflammatory changes in the tympanic cavity are usually accompanied by malaise, fever, deafness, and pain in and about the ear, in the mastoid region, etc.

The onset of acute otitis media in children is often accompanied by a high fever of up to 39-40° C which is sometimes present prior to any local symptoms and is the only sign of the incipient disease.

Functional examination of hearing usually reveals deafness of the conduction type; signs of improvement in hearing in most cases indicate a trend to recovery.

Course. The course of acute otitis media shows wide variations. Mild inflammations without drum perforation

may take a reverse course at any stage and in a very short period end in complete recovery. The duration of the disease taking a purulent form with drum perforation is 2 to 3 weeks in mild cases and up to 4 to 6 weeks in moderate and severe forms. Drum perforation or paracentesis is usually followed by a marked improvement in the patient's general condition: body temperature sometimes drops to normal, severe shooting pains usually decrease, and in general the patient feels much better. Continued high fever and pain felt even after drum perforation may indicate the advent of a complication. Discharge from the ear may vary; it may be scanty, or sometimes so abundant as to suggest a lesion of the mastoid cellular system.

In acute otitis, the perforation is usually seen as a fleck located in most cases in the anteroinferior quadrant and often hidden from sight by the edematous swelling of the drum layers; in such cases it is identified by a light reflex on a pus drop pulsating through the perforation.

At the outset, acute otitis produces tenderness over the bone of the mastoid process which reacts with considerable pain to pressure applied behind the ear. In uncomplicated cases of otitis this tenderness usually disappears after drum perforation. If it is still apparent later, in the third or fourth week of the disease, it is a symptom of the onset of mastoiditis.

Outcome. The most common outcome is full recovery, cessation of pus discharge, healing of the perforation and full restoration of hearing. In other cases, the perforation may remain despite the cessation of pus discharge, and relapsing otitis media may follow reinfection. Finally, otitis which develops after infectious diseases, often takes a chronic course with continued pus discharge and a stubborn perforation. Furthermore, some cases of acute otitis are complicated by mastoiditis. It is also necessary to bear in mind possible intracranial complications of acute otitis, such as sinus thrombosis and pyemia, brain abscess and purulent meningitis. Therefore, any prognosis in acute suppurative otitis should be made with caution.

Diagnosis. The diagnosis of acute otitis media in its first stage when the drum is not ruptured is based on a typical history, such as a feeling of fullness and shooting pain

in the ear, as well as fever, and on otoscopical examination of the drum. These symptoms and the appearance of pus discharge give ample evidence for establishing a diagnosis of acute suppurative otitis.

In case of concomitant external otitis and narrowing of the auditory meatus, visual inspection of the drum is almost impossible, and a diagnosis of acute otitis media rests on the following data.

External otitis is not accompanied by a considerable loss of hearing as is the case in acute otitis media, and the greatest tenderness occurs in the tragus and not in the mastoid process in contrast to otitis media. An admixture of mucus in pus discharge will surely indicate the presence of otitis media.

It is often very difficult to make a diagnosis in infants owing to the lack of co-operation on the part of the small patient. Otoscopy and careful general examination usually solve the problem.

Treatment. General treatment is to confine the patient to bed and ensure him full rest; at the same time he is given aspirin, pyramidon and sulfonamides orally. Sharp pains and insomnia may require the administration of soporifics, such as veronal and morphine. In cases of shooting pains and marked redness of the drum, phenol-glycerol ear drops should be used.

Rp. Acidi carbolici 0.3-0.5
Glycerini puri 10.0
MDS. A warm dose of ten drops to be instilled in the ear three times daily and allowed to remain for ten minutes at a time

After this the drops should be dried with cotton wool to avoid irritation of the meatal skin. Phenol-glycerol treatment usually lasts two to three days from the onset of the disease. If pus has appeared these drops must not be used in view of the possibility of burns occurring from carbolic acid released when phenol-glycerol dissolves in the purulent fluid. In aggravations of the inflammatory process, particularly when there is irritation in the mastoid process, cold treatment should be applied in the early days of the inflammation. An ice-bag is wrapped in a doubled towel

and placed on the mastoid process at intervals for several hours daily.

The cold will cause a reflectory spasm of the vessels with limitation of the acute inflammation and often abortion of the incipient inflammation in the mastoid process. The analgetic effect of cold is more pronounced than that of a hot compress. Nevertheless, the later treatment, or treatment in case of intolerance of cold, is by heat, by the application to the ear of a compress, a hot-water bottle, or heat from a blue or "sollux" lamp. On the very first day of the disease vasoconstrictive and antiseptic drops should be instilled into the nose, such as 3% ephedrine solution and 1-2% protargol solution.

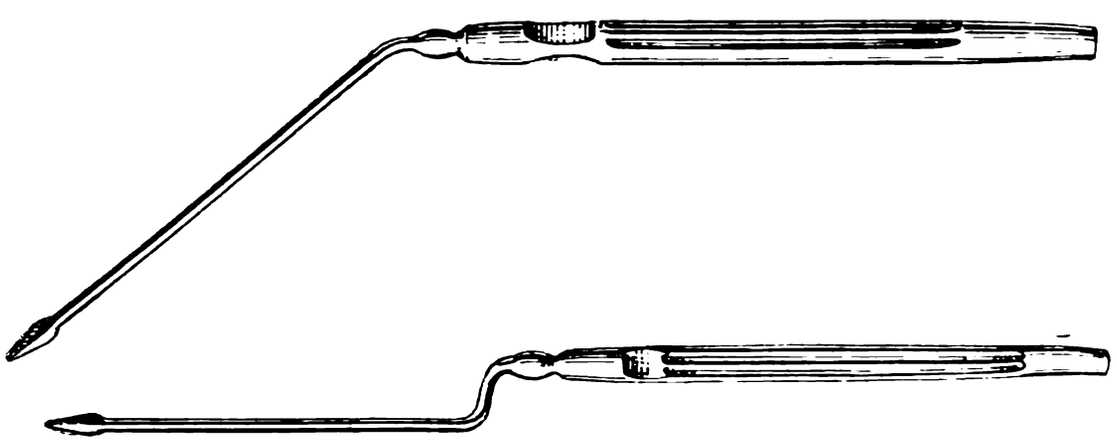


Fig. 22. Myringotomy Knives

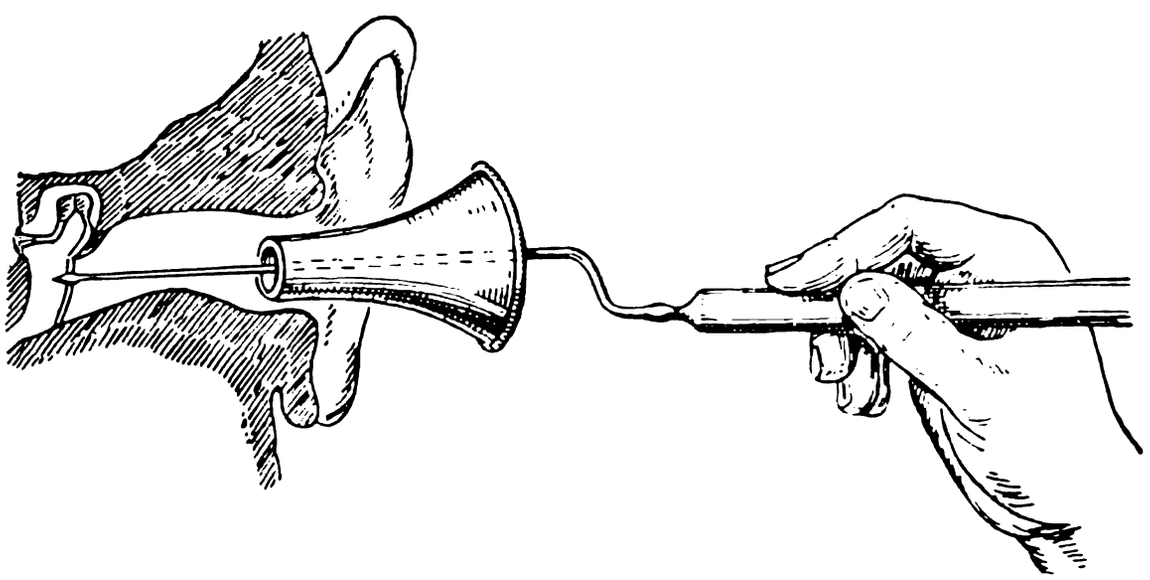


Fig. 23. Paracentesis of Drum

If the inflammatory symptoms do not subside after two to three days of such treatment and the fever remains high, the drum should be incised to release pus and avoid complications. In considering indications for paracentesis the patient's general condition is of paramount importance, while the duration of the disease is of lesser significance. In case of incessant severe pain in the ear and a high fever of 38°C and upwards, paracentesis is imperative immediately, even if hyperemia of the drum is unmarked. The range of recognised indications for paracentesis should be broadened so that it may be performed even where there has been penicillin and other antibiotic treatment, if a considerable hearing loss has been noted in a lingering or torpid course of otitis. It is preferable that the operation of paracentesis (myringotomy) be entrusted to a specialist, though it can be made by any physician or medical assistant with adequate experience in otoscopy.

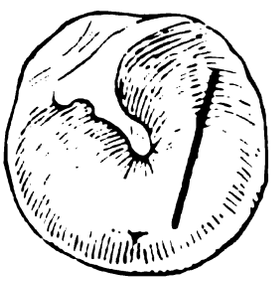


Fig. 24. Site of Incision of Drum

Acute otitis media occurring in the presence of infectious diseases, such as measles, influenza and scarlet fever, requires early paracentesis irrespective of when the disease started. Symptoms of meningeal irritation often observed in small children are undoubted indications for immediate myringotomy. The incision is made on the drum bulge, well-lit and kept under direct observation, with a paracentetic needle (Fig. 22), otherwise known as myringotomy knife, and carried downwards in the posterior quadrants of the drum (Fig. 23-24). Usually seropurulent or purulent discharge appears immediately or shortly after the operation with a decrease in the drum bulge and tension (coloured Table I, Fig. 2b). The aftercare is to ensure free pus drainage from the middle ear cavities. This is achieved by insertion of strips of sterile gauze into the auditory canal until they come into contact with the drum (Fig. 25). At first these ear tampons should be changed six to eight times a day, and later only three to four times a day according to the decrease in pus discharge.

Abundant or thick viscous exudate may be washed out by an ear douche with a warm 4% boric acid solution.

The canal should then be dried with cotton wool wrapped on a slender probe. The ear may be syringed once or twice daily in accordance with the appropriate rules. The temperature of the solution should be about 37°C; the douche should be made with a rubber bulb directing the stream under low

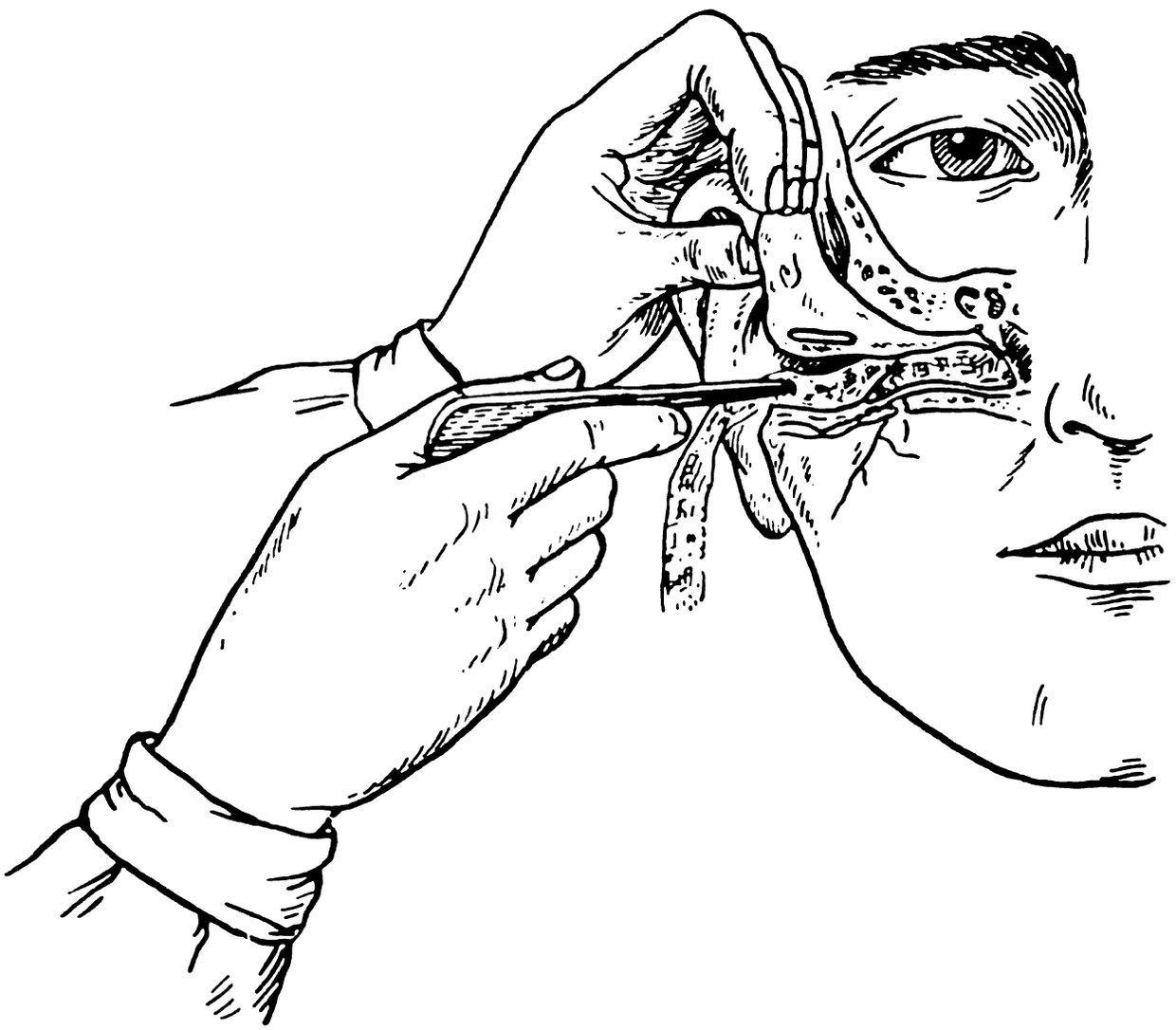


Fig. 25. Insertion of Gauze Strip into Meatus

pressure along the posterior wall of the auditory meatus with the pinna being drawn upwards and backwards.

Pus congestion in the external auditory meatus may cause inflammation and swelling of its skin lining, which in turn will obstruct pus drainage from the middle ear.

Following paracentesis, body temperature falls at once or drops gradually, as in influenzal otitis, and the pain is relieved with an attendant amelioration of the patient's general condition.

The discharge which at first is copious and purulent gradually decreases and passes to a seropurulent and then mucous condition before finally disappearing. The drum perforation heals, the hearing shows signs of improvement and returns to normal. After the drum has healed, the Eustachian tube may in some cases have to be inflated to improve the hearing.

Cases of stubborn perforation in acute otitis media or of it becoming chronic are now much fewer than before, and these mostly occur in otitis following infectious diseases. Apart from preventive measures to improve the health of the population, new sulfonamide and antibiotic drugs have done much to keep down the rate of acute otitis of a severe type.

The above-described treatment is usually sufficient for young and middle-aged patients to recover from acute otitis with moderate symptoms.

Patients with concurrent diseases, small children and subjects with a severe form of acute otitis concurrent with an infectious disease should be treated with sulfonamide and antibiotic drugs to prevent possible complications. Streptocide and sulfadimezin are given to adults in a 0.5-1.0 g dose to be taken orally four to six times in 24 hours for five to six days until obvious signs indicate that all symptoms of acute otitis have abated.

Penicillin and streptomycin should be given in ear drops and intramuscular injections. The auditory meatus should be cleansed of pus and filled with penicillin or streptomycin solution in a dosage of 10,000 to 50,000 units per ml. By gentle pressure on the tragus one should try and force the solution into the tympanic cavity and Eustachian tube.

A combined use of local and intramuscular administrations of antibiotics has proved more effective. Adult patients are given intramuscular injection of penicillin in a dose of 100,000 to 200,000 units to be repeated at regular intervals five to six times in 24 hours. Six, eight or ten days of such treatment are enough to abort the process. The symptoms of recovery are an amelioration of general condition, cessation of pus discharge on the second or third day in case of correct treatment, the healing of the drum on the third to fourth day, and restoration of hearing. A favourable outcome, restoration of hearing in particular, is facilitated by an

early ear, or rather, tubal inflation which should be carried out immediately after the inflammatory symptoms have abated, and pus discharge has ceased; if necessary this should be preceded by decongestion and cleansing of the nasal cavity. The masking effect of antibiotics must be taken into account.

Patients who have undergone penicillin and streptomycin treatment should remain under medical observation even after all symptoms of acute otitis media have disappeared. Cessation of nearly all of such symptoms, excluding hearing loss, and the patient's satisfactory general condition in the first three to four days of antibiotic treatment are often mistaken for an indication that this treatment is no longer required, which may lead to a relapse, sometimes accompanied by serious complications.

Prophylaxis. Prevention of acute otitis media consists in control of infectious diseases, as well as acute and chronic diseases of the nose and nasopharynx. Early treatment of acute nasal and nasopharyngeal lesions combined with hardening the entire body serves to reduce the incidence of inflammations in the middle ear.

Acute Otitis Media in Infants and Young Children

The rate of acute otitis in infancy and early childhood is much higher than in adult age. In children, particularly in the first months of life, unlike adults, the functional and morphological peculiarities of the central nervous system account for their imperfect adaptability to changes in the external environment. Among the causes of this predisposition are above all ailments weakening general body resistance. In infants-in-arms these are alimentary and metabolic disturbances, in older children—infectious diseases, such as scarlet fever, measles, and influenza. The higher incidence of middle ear diseases in infants is usually regarded as linked with the remnants of embryonic myxoid tissue filling the middle ear cavity and most susceptible to suppuration caused by the entrance of infectious germs through the Eustachian tube, which is wider and shorter than in adults. The infant's constant lying on the back and lack of the expectoration reflex, which might have enabled him to clear his

throat in acute nasal and nasopharyngeal diseases, make it easy for the germs to enter the Eustachian tube. Adenoid hyperplasias and inflammations of the pharyngeal tonsil (adenoiditis) often serve as a cause of relapsing otitis in older children.

Symptoms. Acute otitis in infants often develops unnoticed until the appearance of pus discharge from the ear. In a mild case the baby is restless without obvious reason, in a more severe, particularly suppurative form, it cries and screams, and its sleep is broken. The baby rolls its head, rubs its sick ear against the pillow, and pulls at the ear, it refuses the breast or takes it only to drop it again, because sucking and swallowing give it added pain. The baby is less restless when lying on the side of the sick ear, because it gets warm in this position.

Because of the anatomical and morphological features of the middle ear, the drum membrane may change very little even in the event of suppurative inflammation. The first signs are vanishing of the light reflex, loss of lustre by the drum owing to the maceration of the epidermis, and hyperemia along the handle of the malleus. Later, the drum may lose its outline and form a bulge. It should be borne in mind that hyperemia in babies may be caused by their violent screaming. This symptom, therefore, without other signs of inflammation is not always indicative of a midde ear disease.

A major symptom is pain on pressure on the tragus as well as the rapid onset of fever of and above 40 °C. Children with hypotrophy, rickets and other serious diseases, however, may develop acute otitis with a slight fever or no fever at all. In severe cases meningeal symptoms are to be found, such as stiff neck, vomiting, coma, and convulsions, known collectively as meningism. These quickly subside following paracentesis or a spontaneous drum perforation. Acute otitis in breast-fed babies often simulates intestinal intoxication attended with continuous vomiting, diarrhea, loss in weight, etc. This may distract one's attention from the real cause, otitis. Only the subsequent perforation of the drum, the appearance of pus discharge and a progressive amelioration of the child's general condition will reveal the genuine cause of intoxication.

Course. Basically the course of otitis depends on the child's general condition. In children with a constitutional disturbance provoked by hypotrophy, rickets, and exudative diathesis the disease may be protracted and may produce relapses and complications. In mild cases recovery follows in seven to ten days. Marked otitis in children passes through the same stages as in adults. Spontaneous pus discharge occurs at a comparatively later date. Otitis following infectious diseases, such as scarlet fever, measles, and influenza, runs a particularly unfavourable course and extensive destruction of the drum may rapidly ensue.

Since acute otitis in infants may sometimes show no adequate symptoms, it is often difficult to diagnose. If the child has a high fever or his general condition is grave, in the absence of symptoms of a general disease one should always resort to otoscopy.

Treatment. Acute otitis in children is managed in the same way as in adults. It should be borne in mind that in children spontaneous pus discharge may come with a delay, while the subjective symptoms may be at variance with the gravity of the morbid condition in the ear. Therefore, indications for paracentesis should be considered in regard to the patient's general condition remembering that insignificant changes in the drum membrane are often indicative of a suppurative inflammation.

Hot compresses with liquid petrolatum should be applied to the ear of small children with acute otitis, as alcohol compresses cause skin irritation. Physiotherapeutic treatment is also recommended, such as heat from a blue or "sollux" lamp. Nasal drop instillations of 3% boric acid solution mixed with adrenalin (1 drop of adrenalin per ml of solution) should also be instituted. Following drum perforation drops of 0.5% zinc solution are instilled into the diseased ear.

Frequent and careful cleansing of the external auditory canal is required to prevent external otitis. In grave cases the most effective remedy is intramuscular penicillin injections of 100,000 unit doses repeated at regular intervals up to 6-8 times in 24 hours for a period of five to six days.

Prophylaxis. Prevention of acute otitis in children consists in general hardening of the body and strengthening of

body resistance. Special attention should be given to the treatment of inflammatory processes in the nose and nasopharynx and prevention of infectious diseases.

Acute Otitis Media in Infectious Diseases

One of the most common complications occurring in many acute infectious diseases are acute, often purulent, inflammations of the middle ear. Until recently, the rate of middle ear affections in acute infectious diseases was very high, particularly among children. Such diseases include scarlet fever, measles, influenza, typhoids and diphtheria. For instance, the incidence of suppurative otitis during some scarlet fever epidemics was between 17 and 27 per cent. Today, the incidence of scarlet fever otitis in children's hospitals has been reduced to fractions of a per cent owing to a number of prophylactic measures, such as a nearly 100 per cent hospitalisation of contagious patients, prophylactic examination by specialists and timely antibiotic treatment.

Nevertheless, there are isolated cases of acute suppurative otitis, some taking a severe course, in subjects with a weakened body resistance or concurrent infectious diseases.

Acute otitis caused by infectious diseases is called secondary in contradistinction to primary, genuine otitis occurring in healthy subjects. Acute otitis appearing at the very onset of an infectious disease should be regarded as a local symptom of a constitutional disturbance spreading by the hematogenous way, that is through the blood; on the other hand, otitis following marked inflammatory symptoms in the pharynx, as in necrotic angina, will undoubtedly have been caused by infectious germs penetrating through the Eustachian tube.

Until recently, most cases of otitis caused by an infectious disease in consequence of a disturbed body resistance had a more severe course than that of ordinary otitis; usually there was no full recovery, and the ear disease often took a chronic course with stubborn aplasia of the drum perforation, constant pus discharge and impaired hearing.

This kind of otitis is known to have two forms, one which

does not differ from ordinary otitis, the other, more severe form, presenting some typical features peculiar to any infectious disease.

Symptoms of necrotic otitis often produced by scarlet fever may occur in the early days of the latter disease if its course is severe. Together with necrotic otitis a necrotic inflammation of the pharynx and nasopharynx is usually present. Because of the fever and grave general condition the onset of otitis in scarlet fever may pass unnoticed, and sometimes only pus discharge will indicate complication. The inflammatory symptoms resulting from the onset of vascular thrombosis in the mucosa are accompanied by an increasingly extensive destruction of the drum and the ossicles, and often produce necrosis in the bony walls of the tympanic cavity with their subsequent sequestration. Not infrequently, as a result of sequestration, the suppuration extends to the internal ear causing labyrinthine symptoms of vertigo, nausea and vomiting, and culminating in complete deafness. Already at the beginning of the disease the purulent discharge has a foul odour. Extension of necrosis to the Fallopian canal is followed by facial paralysis.

Acute otitis in an infectious disease often produces mastoiditis, and is a most frequent cause of chronic suppurative otitis. Cases of recovery from necrotic otitis with complete restoration of hearing are almost nonexistent.

Today, antibiotic treatment of scarlet fever has drastically reduced the incidence and severity of its complications, including otitis.

Acute otitis in measles is a very frequent occurrence, and its course is, as a rule, milder than that of scarlet fever otitis.

Emaciated subjects, however, as well as those with concurrent infections may develop necrotic forms of measles otitis marked by severe symptoms.

Influenzal forms of otitis are characterised by hemorrhagic exudation on the drum and in the bony portion of the auditory meatus, often seen as small blebs with bloody contents. Following perforation of the drum the sanguineous discharge changes to pus in a few days' time. The severe neuralgic pain in the ear and corresponding half of the

head is not relieved at once, even after drum perforation.

There may be hemorrhages in the labyrinth and the auditory nerve trunk similar to the punctate hemorrhage in the drum membrane. A toxic affection of the acoustic nerve sometimes causes severe hearing loss.

Treatment. Treatment of otitis caused by an infectious disease is in most cases by intramuscular injections of penicillin in adequate doses, the administration of which should be continued even after the first signs of improvement appear.

In case of drum perforation and pus discharge local treatment is the same as in ordinary otitis. Prior to penicillin treatment these forms of otitis were more often complicated by mastoiditis which demanded surgical interference.

Today, the wide prophylactic use of penicillin in scarlet fever and measles has brought the rate of their suppurative otitic complications down to one per cent.

Prophylaxis. Prophylactic measures for patients with acute infectious diseases, apart from their early isolation, include careful general attendance and preventive treatment of the nose and throat with antiseptic gargles, instillation of penicillin drops into the nose, etc.

Mastoiditis

An acute inflammation of the mastoid cellular system is among the most frequent complications of acute otitis. Any acute inflammation of the tympanic cavity with more or less pronounced symptoms usually extends to the mastoid process, owing to the close relation of the tympanic cavity with the antrum, and through the latter, with the pneumatic cells of the mastoid process. The body reaction may later reverse the process both in the tympanic cavity and the mastoid cells and ensure complete recovery.

The development of mastoiditis, as of other pathological processes of infectious origin, depends not so much on the virulence of the infection as on the reaction of the body to the latter, that is on the general condition and reactivity of the body.

In cases of otitis following acute infectious diseases, as well as in subjects with a reduced body resistance the morbid process may fail to take a reverse course, and recovery will not follow. Moreover, the inflammatory process may involve bone tissue, break down the thin walls between pneumatic cells of the mastoid process and form one large cavity filled with pus and granulations. Further destruction of the inner cellular system may produce a fistula or a subperiosteal abscess on the outer surface of the mastoid process. Destruction of the inner and upper bony walls of the mastoid cells often leads to intracranial complications, such as thrombosis of the sigmoid venous sinus, abscess in the brain or cerebellum, and meningitis.

Mastoiditis usually develops by the early third and late fourth week of acute otitis. These periods, however, are not absolute and may vary in any direction.

Symptoms and course. One of the outstanding signs of mastoiditis is pain in the mastoid area felt on the third to fourth week of the onset of acute otitis. Often spontaneous, this pain sometimes troubles the patient at night and increases on pressure applied to the mastoid tip and plane (*planum mastoideum*). An important sign of mastoiditis is narrowing of the external auditory meatus caused by swelling of the posterosuperior wall in its bony portion, as well as edema of the overlying skin and tissue of the mastoid process and formation of a subperiosteal abscess. The latter is preceded by inflammatory infiltration, erythema and pasty softness in the mastoid skin. The retroauricular abscess is indicated by fluctuation and loss of the skin fold behind the auricle with the latter being displaced forward and downwards (Fig. 26).

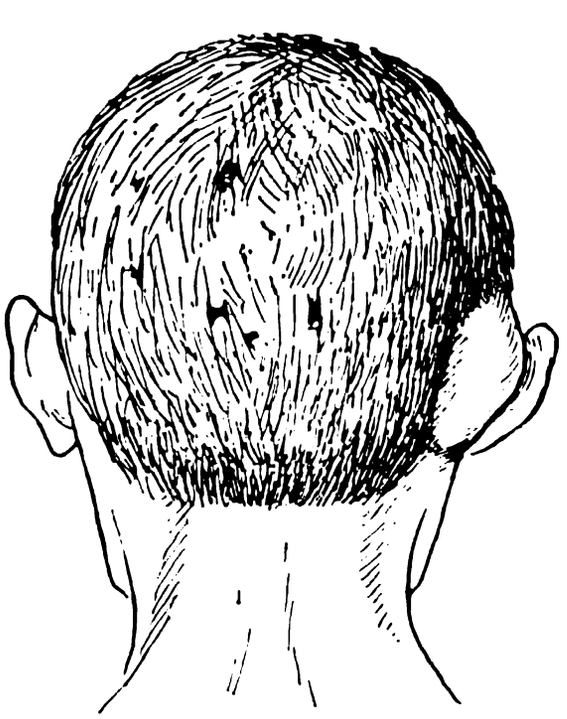


Fig. 26. Swelling behind the Ear in Typical Mastoiditis

The bursting of the subperiosteal abscess gives rise to

a skin fistula usually leading into the antrum. In childhood when connection between separate parts of the temporal bone is incomplete, swelling and a retroauricular abscess are a more frequent occurrence. Cases of this abscess are much rarer in adult and senile subjects who have a much harder cortical layer. Special varieties of mastoiditis may also develop depending on the character of mastoid pneumatisation and preferential suppuration in a specific group of cells. In suppuration of the apical cells the pus may break through the inner wall of the mastoid tip and spread downwards under the sternocleidomastoid muscle. Then, a heavy painful swelling will extend downwards from the apex of the mastoid process over the side of the neck (apical or Bezold's mastoiditis). Suppuration of a group of cells at the root of the zygomatic process may cause swelling and later an abscess in front of, and above, the auricle (zygomatic mastoiditis).

The patient usually feels ill; complains of headache, insomnia, lack of appetite, etc. The temperature, which fell after the perforation, rises again sometimes up to 38.5°C, but in many cases may remain subfebrile. The drum remains congested and swollen for a long time, the discharge from the ear increases and turns into thick frank pus.

Diagnosis. It is comparatively easy to make a diagnosis of mastoiditis in the presence of external symptoms such as a subperiosteal abscess. The most important diagnostic sign is sagging of the posterosuperior wall of the auditory meatus. This wall is at the same time the anteroinferior wall of the mastoid antrum. In extensive pneumatisation it will be thin, and in suppurations of the mastoid process it will very soon be swollen in the periosteum. The presence of acute tenderness over the mastoid process on pressure behind the auditory meatus (place of antrum projection) is also a characteristic sign of mastoiditis. There is usually conduction deafness.

The progress of mastoiditis is sometimes accompanied by a few mild symptoms. These atypical or latent forms of mastoiditis often develop at normal body temperature, with the drum intact, but always involving loss of hearing acuity. Despite mild symptoms bone lesions in such cases may be considerable, particularly in deep regions border-

ing on the cranial cavity. These forms of mastoiditis often produce intracranial complications. A careful analysis of all the patient's complaints and the dynamics of the morbid process, as well as a thorough physical examination with the use of auxiliary methods, is helpful in establishing a timely diagnosis. Among such methods is X-ray examination, which helps to determine the type of the cellular system, as well as the scope and degree of bone destruction in the mastoid process.

Blood changes in mastoiditis, just as in other acute purulent diseases, are often characterised by marked leukocytosis (up to 10,000-12,000 per cu mm of blood), a leftward shift in the white cell count, a decrease in number or total disappearance of eosinophils, a high E.S.R., etc.

Prophylaxis. Mastoiditis prevention consists in a correct and timely treatment of acute suppurative otitis media.

Treatment. Mastoiditis should at first be treated conservatively. Should this fail, the diagnosis must be confirmed before proceeding to surgical interference.

Conservative treatment consists in prolonging the treatment given for acute otitis. Measures to ensure free pus drainage and application of cold (ice) to the mastoid region at the onset of the disease, as well as penicillin treatment and administration of oral sulfonamides may all have a favourable effect on the morbid process. Yet despite these measures surgical treatment is often necessary. As a rule, it is difficult to set a fixed date for the operation. An operation is usually indicated if six weeks after the onset of acute otitis the pus discharge continues, while symptoms of mastoiditis persist and even tend to aggravate. An operation may be necessary even a few days after the onset of otitis should symptoms of intracranial complications appear.

Today the wide application of antibiotics in the treatment of otitis has made mastoiditis a rare occurrence. Moreover, many cases of mastoiditis which formerly required surgical interference can now be cured by protracted treatment with penicillin or streptomycin in large doses. Penicillin is given by intramuscular injections in 100,000 unit doses repeated six to eight times in 24 hours for a period of 10, 12 or 14 days. Signs of recovery are improvement

in the general condition, cessation of pus discharge and healing of the drum perforation, as well as a progressive restoration of hearing. The inflation of the tube should be made as early as possible.

Recovery is no guarantee against a possible relapse. Therefore, the convalescent should be kept under observation for a month or two with an occasional X-ray check-up should need arise.

The operation on the mastoid process, known as mastoidectomy, is performed under local and sometimes under general anesthesia. A curved incision is made 1 cm behind and following the attachment of the auricle. The incision extends from a point on a level with the upper margin of the pinna to the mastoid tip. The lips of the wound are drawn apart with retractors to keep the mastoid surface open for examination. Should a fistula or darkened and soft portions of bone be discovered, the operation must be started at this place. An eyed probe is inserted into the fistula to find a direction along which to dig into bone, as in nearly all cases this fistula will lead into the mastoid antrum. Should a fistula be absent, the operation must be started in a typical place determined by landmarks. The upper border of the operative area is the temporal line which is a backward extension of the upper edge of the zygomatic process; above this line lies the floor of the middle cranial fossa; the anterior border is the spine above the external auditory meatus and the latter's posterior wall, which must not be gouged off lest the auditory meatus should become narrow later on. Trephination is begun by attacking the bone right behind the spine on the *planum mastoidenum*. After the process has been opened pus may flow in quantity to reveal a cavity filled with loose granulations which are easy to remove with a curette down to the antrum.

All the carious and soft bone should be removed carefully until the antrum has been exposed. Probing of the antrum resulting in the appearance of air bubbles is an aid to establish whether or not the antrum has been opened. A thin curved probe easily passes forward through the antrum into the epitympanic recess being accompanied by slight pain. The antrum is then widened somewhat with a small

curette, and the granulations are thoroughly scraped out with utmost care. In conclusion it is necessary to examine every detail of the operative cavity (Fig. 27), and to smooth out with a gouge and forceps all bulging and rough bone parts, as well as to remove all remaining granulations and necrotic portions of carious and soft bone down to normal bone which is easily identified by its hard consistency.

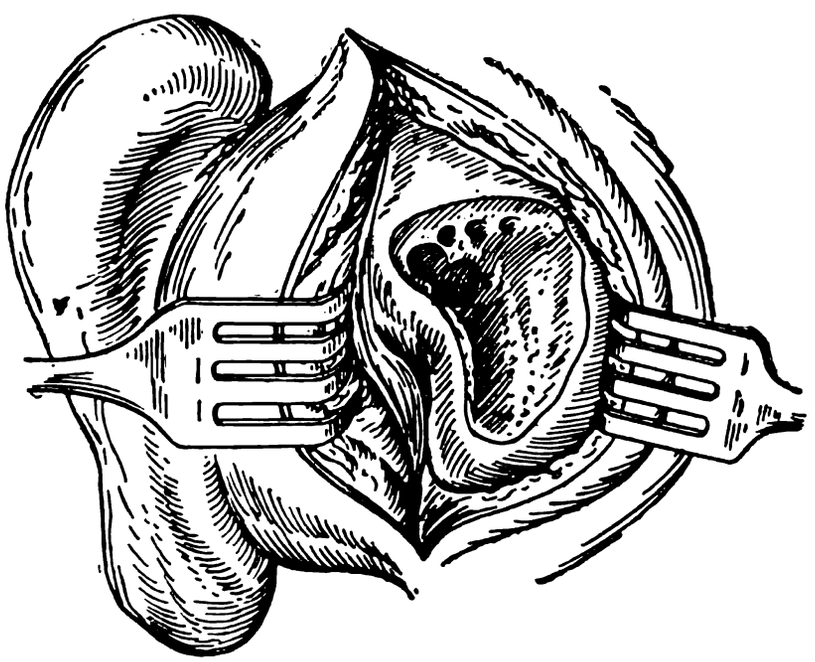


Fig. 27. Opening of Mastoid Process (Antrotomy)

Care must be taken in opening the mastoid process to avoid injury to the sigmoid venous sinus, the dura mater, the middle cranial fossa, the facial nerve and the external semicircular canal.

Postoperative treatment. The operation is usually concluded by filling the wound with sulfonamide or antibiotic powder (penicillin, streptomycin) and packing it lightly with tampons. If there is no pain in the wound and no fever, the dry dressing and packing are changed for the first time after six or seven days at the earliest. Later dressings are applied every day or every other day until the cavity is filled with granulations, and a thick scar covered with epidermis forms. Pus discharge from the ear usually ceases within a few days after the operation; the drum perforation heals, and the hearing returns to normal.

Recently, a method of closing the trephination wound with stitches has been used leaving a slender drain in the antrum. The open end of the drain which exits above the dressing serves to instil into the wound penicillin solution in 2 ml doses twice a day (5,000 units per ml). The trephination wound usually heals by first intention in seven to ten days, which serves to shorten the patient's stay in hospital and quickly restores his work capacity.

Postoperative care. It is exceedingly important to give the surgical patient complete rest and medical care in the pre- and postoperative periods. Therefore, the role of the hospital nurse in providing this care for patients with ear diseases and complications can hardly be overestimated. The nurse should ensure the patient full rest, quietness and comfort and see to it that he has regular meals and stool. She should take care that he sleeps peacefully and on instructions from the doctor give him analgetic, soporific and sedative drugs favourably affecting the central nervous system.

Should such symptoms as a severe headache, a sharp rise in body temperature, a chill, a rise or drop in pulse rate, nausea and vomiting appear, it is the duty of the nurse to report them immediately to the doctor in charge.

Chronic Suppurative Otitis Media

(*Otitis media purulenta chronica*)

The following three symptoms are the permanent characteristics of chronic suppurative otitis media: a stubborn perforation of the drum, incessant or intermittent pus discharge from the ear, and deafness of varying degrees.

The causes of an acute suppurative otitis media taking a chronic course may be of general and local origin.

Among general causes are acute infectious diseases producing deep necrotic changes in the middle ear already at the very beginning of acute otitis. Among them is otitis of the necrotic type seen with scarlet fever, measles and diphtheria. Otitis occurring in anemic and emaciated patients with grave general diseases, as tuberculosis, diabetes, acute alimentary disturbances, etc., often turns chronic.

Local causes may include all chronic inflammations of the nose and nasopharynx, such as adenoids, chronic coryza,

chronic purulent inflammations of the paranasal sinuses, which maintain suppuration in the tympanic cavity as a result of continuous infection through the Eustachian tube. Incorrect and inadequate treatment of acute otitis may sometimes contribute to its transition into a chronic form.

Chronic suppurative otitis media may persist for decades without much trouble to the patient, because in the absence of complications it causes no pain or fever; in a monaural case, the patient may even fail to notice a certain loss of hearing.

The patient usually applies for medical aid with complaints of an increased pus discharge, which is due to an aggravation of the chronic course, as well as of a foul smell from the ear or signs of intracranial complications; in a binaural case, the major complaint is impaired hearing.

For their clinical course and prognosis, cases of chronic suppurative otitis are divided into two forms: *otitis with a central perforation* and *otitis with a marginal perforation* of the drum.

The central perforation is a rupture surrounded on all sides by an intact margin of the drum. The perforation is called marginal if it extends to the bony portion of the auditory meatus or is located in Shrapnell's membrane. The perforation may be round, elliptical or bean-shaped and vary in size.

As only the mucosa is involved in the inflammatory process in otitis with a central perforation (coloured Table II, Figs. 1, 2, 3) the latter is sometimes called benign or uncomplicated. Otitis with a marginal perforation, which besides the mucosa affects the bony walls, is known as the dangerous, complicated type.

Chronic suppurative otitis with a marginal perforation is certainly a more serious disease. Marginal perforations usually occur in the posteroanterior quadrant of the drum or in the pars flaccida of Shrapnell.

The perforation may be in punctures over the Shrapnell membrane, or it may be so extensive as to leave no trace of the drum. The inflammation, as is shown by the drum perforation, primarily invades the upper portions of the

tympanic cavity causing a carious destruction of the external wall of the epitympanic recess (*epitympanum*). Therefore, this kind of otitis is also called epitympanitis, as distinct from the uncomplicated otitis known as mesotympanitis.

The carious process produces complications commonly in the form of granulations, polyps and cholesteatoma (coloured Table II, Fig. 4).

Cholesteatoma is a product of the epitympanic type of chronic suppurative otitis media. It appears in the event of a marginal drum perforation. Through a lesion in the drum margin the epidermis of the external auditory canal forms an outgrowth into the epitympanic recess and the mastoid antrum. Constant desquamation of the horny cells and their accumulation in the middle ear cavities serves to build up a compact white mass known as cholesteatoma. This mass tends to grow infinitely and its pressure may break down the surrounding bony walls. This destruction may extend outwards, towards the mastoid process, and inwards, into the cranial cavity down to the dura mater, the middle and posterior cranial fossae, the sigmoid sinus, as well as to the wall of the external semicircular canal and the facial nerve. In an aggravated middle otitis, cholesteatoma is liable to putrefaction and is the most frequent cause of intracranial complications.

Diagnosis. Otoscopy and probing are the common means to detect chronic suppurative otitis media and find out whether it is in the simple or complicated form. Examination of the ear should be preceded by a meticulous cleansing of the auditory canal with a douche or dry application to remove pus. *It is very important* to locate the place of drum perforation and *determine whether it is* of the central or the marginal type. It is sometimes hard to detect a small perforation in Shrapnell's membrane as it may be hidden under a crust of dry pus. Extensive or total destruction of the drum on the contrary reveals changes in the middle ear mucosa which is usually swollen and red, and is covered with granulations appearing as tiny reddish grains bleeding readily if touched with a probe. Bare bone, often with a rough surface, is felt in a probe examination.

The amount and character of pus discharge may also prove essential for estimating the gravity of the process. An odourless discharge of thick frank pus is a characteristic sign of affection of the middle ear mucosa alone (mesotympanitis). An admixture of mucous threads will point to the Eustachian tube being envolved in the inflammatory process (tubomesotympanitis). A scanty discharge with a blood admixture and a stubborn rank smell which persists despite medical treatment will be proof of an insidious process of bone necrosis in the middle ear (epitympanitis).

Inadequate treatment of the diseased ear will be followed by pus congestion in the middle ear cavity and its decomposition by saprophytic germs, which will lend the discharge a disagreeable odour even in innocent forms of otitis. In such cases the odour disappears after two to three weeks of treatment, which is not likely to happen in a necrotic process. All forms of otitis are accompanied by deafness of the conduction type. Epitympanitis caused by a toxic affection of the labyrinth may produce a mixed hearing loss involving nerve deafness.

Treatment. Treatment of suppurative otitis with a central perforation should be along conservative lines.

In dealing with chronic suppurative otitis special efforts should be made to remove the general causes of predisposition which have made the inflammation take a chronic course. This should be followed by local treatment by means of thorough and periodical removal of pus from the ear and instillation of antiseptic astringents to affect the middle ear mucosa. In the treatment of suppurative otitis it is important to eliminate the inflammation in the nose and nasopharynx. Adenoidectomy, operations for diseases of the paranasal sinuses, as well as removal of hypertrophic posterior ends of the lower conchae, etc., are in many cases likely to abort the suppuration and ensure recovery.

The desired astringent and antiseptic effect is produced by ear drops made of 4% boric acid solution, 3% Burow's solution or 1% zinc sulfate solution.

Rp. Sol. Zinci sulfurici 1% 10.0
DS. Dose of eight drops to be instilled into the ear two to three times daily

The following drops are also helpful:

Rp. Acidi borici 0.8
Hydrogenii hyperoxydati
Spiritus vini rectificati āā 10.0
MDS. Dose of eight drops to be instilled into the ear three times daily

Furacilin is also an efficient antibacterial drug.

Rp. Sol. Furacilini
in Spiritus vini 1:1,500 20.0
DS. Dose of eight drops to be instilled into the ear three times daily

An inflammatory swelling of the middle ear mucosa is treated with 40% to 96% boric alcohol or painted with a 3% silver nitrate stick once or twice a week.

Full recovery with healing of the drum perforation and restoration of hearing occurs rarely; more frequently there is cessation of pus discharge, a stubborn drum perforation, scars, ankylosis of the auditory ossicles and impaired hearing.

Treatment of suppurative otitis with a marginal perforation is more difficult. The presence of a marginal perforation is not necessarily an indication for an operation; yet the latter may be required if conservative treatment fails. In case of free access to the attic and antrum and the absence of polyps, granulations and dense cicatricial adhesions, conservative treatment should be tried even if a small cholesteatoma is present. The ear should be filled with drops of boric or salicylic alcohol following the most meticulous cleansing of all middle ear cavities where access is possible.

Rp. Acidi salicylici 0.2
Spiritus vini rectificati 20.0
MDS. Dose of eight drops to be instilled into the ear three times a day and allowed to remain for 15 to 20 minutes

In some patients alcohol causes a burning sensation and pain in the ear, in which case drops of softer solutions

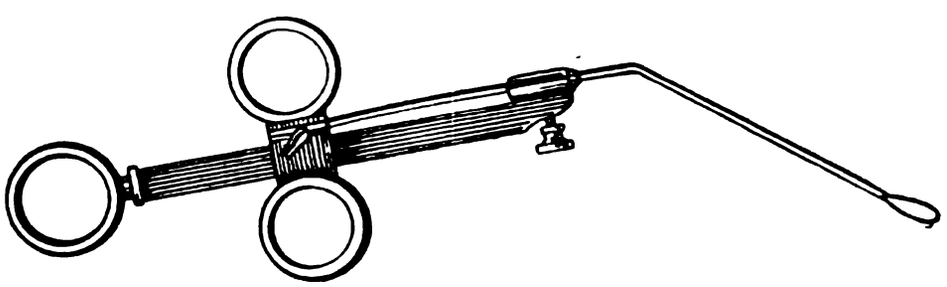


Fig. 28. Aural Snare

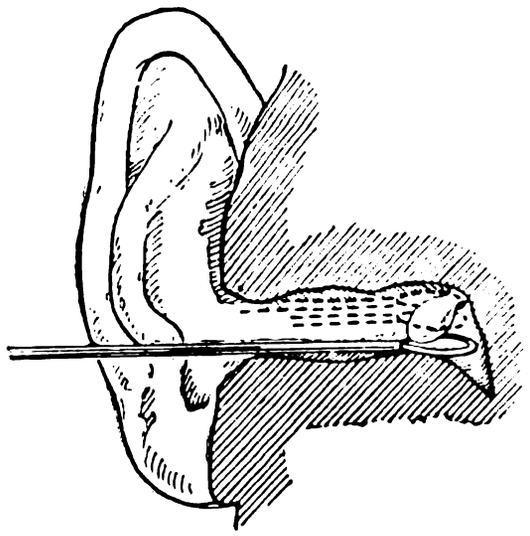


Fig. 29. Removal of a Polyp

should be used at first, such as 40% alcohol. Polyps are removed with an aural snare (Figs. 28 and 29); granulations are cauterised with 10% silver nitrate solution or trichloracetic acid. In a marginal perforation, the suppuration is usually confined to the epitympanic recess. In such cases common ear syringing usually fails to achieve the desired result. Therefore, repeated attic douches of boric alcohol are used through the so-called attic cannula with a curved beak which is introduced through the perforation in the upper part of the drum (Fig. 30).

This treatment combined with careful removal of pus and, occasionally, with application of a powder of very fine boric acid or sulfonamide preparations to the middle ear mucosa serves to remove superficial carious bone and terminate pus discharge, which may be followed by cicatrisation and epidermisation of the middle ear cavities.

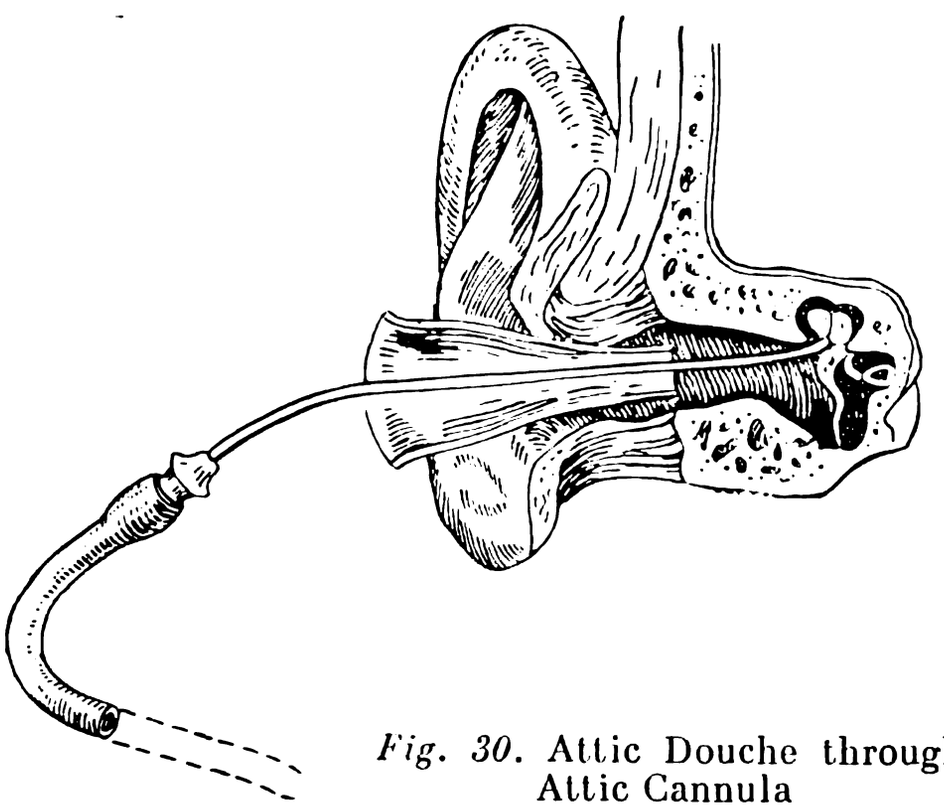


Fig. 30. Attic Douche through Attic Cannula

Should conservative treatment prove unsuccessful, which is usually attributable to the presence of cholesteatoma or an extensive and deep-seated carious process, as well as in cases of persistent headache, attacks of vertigo and fever a radical operation on the middle ear is necessary.

Radical operation on the middle ear is indicated in cholesteatoma and facial nerve paralysis. The operation would be urgently indicated even if the slightest suspicion of an intracranial complication existed, such as sinus thrombosis, purulent meningitis, abscess in the brain or cerebellum.

The *radical operation*. The radical operation essentially consists in the tympanic cavity, the epitympanic recess, the antrum with the remaining mastoid cells and the external auditory meatus being thrown into one wide cavity. Therefore, this operation is also known as radical mastoidectomy. A thorough removal of carious bone and the cholesteatoma will ensure free pus drainage through the auditory canal and prevent possible intracranial complications.

The operation begins with opening the antrum, as in mastoidectomy; next follows the removal of the upper section of the posterior bony wall of the external auditory meatus and the external wall of the attic. Here, in the depth of the operative cavity, great care must be taken to avoid injury to the facial nerve, as the descending knee of the facial nerve canal is located in the depth of the posterior bony wall of the auditory meatus. The concluding stage of the operation is removal of all necrotic auditory ossicles apart from the stapes. Polyps, granulations and carious bone are carefully removed with a curette. The operation is rounded off with a plastic repair in order that the walls of the operative cavity may later be overgrown with epidermis. For this purpose one or two flaps are cut out of the skin of the posterior wall and roof of the external auditory meatus and are transplanted on to the lower or upper parts of the wound. The flaps serve as a source of epidermis for the whole trephination field. The skin wound behind the ear is either sutured or left open if the meninges are exposed. The operation area is packed with a tampon soaked in iodoform or penicillin solution (10,000 units per ml). Dry dressing is first applied on the sixth to eighth day following the operation, provided there is no fever or

pain in the wound. The postoperative treatment is rather complicated and normally continues for at least six to eight weeks. In some cases, tympanoplasty is performed if there is no affection of the middle ear together with signs of an intracranial complication. The aim of this operation is not only to remove pathological tissue from the middle ear but to repair the drum. Not infrequently the hearing also improves as a result of this operation.

Prophylaxis. Chronic suppurative otitis media can be prevented by correct and timely treatment of acute otitis, prophylaxis of the nose and nasopharynx, and general measures to increase body resistance.

Both general and local treatment should be used to cure patients with acute and subacute otitis media until their hearing is fully restored. Speedy recovery from acute otitis is brought about by timely and correct antibiotic treatment with penicillin and streptomycin. The early use of antibiotics is of particular importance in curing acute otitis in children occurring as a complication of acute infectious diseases, such as measles, scarlet fever, etc., because this kind of otitis may produce bone necrosis in the middle ear and often becomes chronic.

In local treatment the need for careful attention to the diseased ear should be emphasised. This includes strict abidance by aseptic rules when cleansing the auditory canal of pus, timely instillation of antiseptic solutions, etc. (see page 37, "General Methods of Nursing and Treatment of Ear Diseases").

Acute otitis often becomes chronic in certain diseases of the nose and nasopharynx obstructing nasal breathing. Among them is hyperplasia of the posterior ends of the nasal conchae, nasal polyps, deformities of the nasal septum, inflammation of the paranasal sinuses, adenoid hyperplasias, etc. The treatment of these diseases by an otorhinolaryngologist leads to restoration of nasal breathing, eliminates obstruction of the Eustachian tube and promotes quick healing in acute inflammations of the middle ear. A reduced body resistance owing to one cause or another may also facilitate acute otitis taking a chronic course, which shows how important it is to conduct general treatment alongside local measures to combat the infection.

In order to prevent exacerbations patients with chronic suppurative otitis media are recommended not to allow water to enter their ears when taking a bath or washing their heads. For this purpose a piece of cotton wool greased in boric petrolatum or some other oil is inserted into the auditory canal. In bad weather with strong wind and dust the patient should protect the auditory canal with clean cotton wool when going outdoors. He must be warned against excessive blowing of the nose through both nostrils at a time, especially during acute or aggravated chronic coryza, as it may cause reinfection and a relapsing inflammation in the ear.

Intracranial complications of chronic suppurative otitis media are prevented by careful treatment and clinical observance of out-patients with epitympanic diseases, such as caries, cholesteatoma and polyps, in order that they may be directed in good time to an E.N.T. hospital for a radical operation on the middle ear.

The medical assistant takes an active part in clinical care of out-patients and selects patients requiring further medical examination.

Labyrinthitis

Inflammation of the internal ear is a very grave complication of suppurative otitis which always involves a severe disturbance of equilibrium, impairment, and sometimes full loss, of hearing in the affected ear. The routes of infection extending from the middle ear into the labyrinth may be through the oval and round windows, as well as through a direct destruction in the labyrinthine bony wall. The latter route is more likely to occur in chronic suppurations in the middle ear complicated by cholesteatoma which gives rise to a fistula in the external semicircular canal. Infectious toxins may also spread into the labyrinth even in case of intact bone and membranes covering the labyrinthine windows. This kind of disease is known as induced labyrinthitis.

According to the clinical picture labyrinthitis is divided into the purulent and serous types.

The purulent type occurs more often in chronic suppu-

rative otitis, and sometimes in cases of acute scarlet fever and influenzal otitis. The destructive effect of cholesteatoma is a particularly frequent cause of labyrinthitis. Purulent labyrinthitis may occur in a localised (circumscribed) and diffuse form.

Symptoms. Labyrinthitis has very typical signs. The disease begins suddenly with the so-called labyrinthine attack, namely, severe giddiness and disturbance in equilibrium followed by fits of nausea and vomiting recurring frequently during several days. The temperature is usually normal, but if there is a considerable rise during a labyrinthine attack this will suggest an incipient inflammation of the meninges.

The serous forms of labyrinthitis cause a drastic deterioration of hearing and of the vestibular function, and in cases of diffuse purulent labyrinthitis both functions are lost. Circumscribed labyrinthitis, however, is not accompanied by total deafness. The nystagmus arising at the very onset of the disease is at first towards the affected ear, and in case of a full failure of the labyrinthine function it changes to the opposite, unaffected side. If there is no onset of meningitis, all disturbances in equilibrium and other symptoms gradually disappear within three to four weeks. Diffuse purulent labyrinthitis results in permanent loss of hearing.

Serous forms of labyrinthitis are in most cases induced by the effect of toxins on the labyrinth through the intact membranes of the round and oval windows. These forms occur in intense inflammations of the middle ear and sometimes may follow an injury made during a radical mastoidectomy.

The clinical signs of serous labyrinthitis will be the same as in the purulent form, though somewhat milder. Recovery from serous labyrinthitis is followed by a partial restoration of hearing.

The most frequent and mild form of labyrinthitis is apparently the so-called *circumscribed labyrinthitis.* In the majority of cases, this form is observed in chronic purulent otitis complicated by cholesteatoma and accompanied by a fistula in the bony capsule of the labyrinth, more often in the area of the external semicircular canal.

The diagnosis of circumscribed labyrinthitis is facilitated by the so-called compression nystagmus which may be evoked in the patient by alternate compression and rarefaction of the air in the auditory canal of the affected ear. When the air is compressed the nystagmus will be towards the affected side, and in case of its rarefaction the nystagmus will reverse in the opposite direction. This kind of nystagmus will indicate a fistula in the external semicircular canal (fistular symptom).

Treatment. If there is no associated intracranial complication, the treatment of labyrinthitis should be conservative. The patient must be strictly confined to bed and kept perfectly quiet. Antibiotic treatment with penicillin and streptomycin should be continued for two to three weeks. Penicillin is given by intramuscular injection in 100,000 to 200,000 unit doses every three hours, or six to eight times in 24 hours on the average. Dosage depends on the gravity of the disease. If there are indications for an operation on the ear, it should be carried out when severe labyrinthine symptoms have subsided, in two to three weeks after the onset of the labyrinthine attack.

In the presence of mastoiditis, a suppurated cholesteatoma or intracranial complications the operation is performed immediately. The mode of surgical interference will depend on the symptoms of the existing complications and the pathological changes discovered at operation.

INTRACRANIAL COMPLICATIONS OF SUPPURATIVE OTITIS

Intracranial complications may develop both in acute and chronic suppurative otitis media, though they occur more frequently in the latter.

There are two types of infection: 1) by contact when as a result of erosion of the bony walls of the tympanic cavity and the mastoid process or labyrinth, the meninges are exposed to the inflammatory focus, or 2) by the blood vessels, when the infection penetrates through them into the cranial cavity.

Intracranial complications occur in the following forms:

Extradural Abscess

(*Abscessus extraduralis*)

An extradural abscess which is a collection of pus between the cranial bone and the dura mater adherent to it is the most frequent intracranial complication. If it overlies the sigmoid sinus, it is called *perisinus* abscess.

Proliferating between the dura mater and the cranial bones the extradural abscess may become very large.

Owing to the inflammation the dura mater becomes thickened and covered with granulations, and if there is free pus drainage through the middle ear it may resist perforation for a long time.

The extradural abscess has no characteristic symptoms, and in most instances is diagnosed at operations undertaken for complications of acute or chronic otitis. The entire affected area of the dura mater is widely exposed during the operation, which is very likely to have a favourable outcome.

Thrombophlebitis and Septicopyemia

(*Thrombophlebitis et septicopyaemia*)

The second type of intracranial complications most frequently met with is thrombophlebitis of the sigmoid sinus, commonly known as lateral sinus thrombosis. Often, erosion of the mastoid cells in mastoiditis and pus accumulation on the sinus walls are an immediate cause of inflammation in the sigmoid sinus walls (phlebitis) followed by formation of a thrombus (clot) in the latter (thrombophlebitis). In time the clot is subject to septic softening, and its infectious particles are swept into the right heart through the internal jugular vein. Metastases in the pulmonary circulation cause abscessed pneumonia, whereas the spread of infection through the general circulation produces metastatic abscesses in the muscles, joints and visceral organs.

The clinical course of the disease presents two major variations, otogenous pyemia and otogenous septicemia, and not infrequently may be of mixed character.

Otogenous pyemia is characterised by a sudden onset which is generally attributed to acute otitis and mastoiditis or to an exacerbation of chronic suppurative otitis follow-

ing the entrance of dirty water into the ear, influenza, etc. The fever, accompanied by a severe rigor and shivering, soon rises as high as 41 °C shortly to fall again, often below normal. A fall in temperature is accompanied by profuse perspiration. These attacks may occur every two days or every day, sometimes two or three times daily and sometimes at regular intervals, similar to malarial attacks. Vomiting and headache may often be among the initial symptoms. As a result of recurrent attacks of pyemic fever and chill the patient steadily loses strength and his face becomes ashen; his pulse rate becomes weak and fast; he suffers from an intestinal disorder, has a furred tongue and bad breath.

Metastases cause infarction and abscesses in the lungs and other organs, which further aggravates the patient's condition.

In septicemia or septic poisoning, the fever is high and fluctuating within narrow limits, while rigors are not severe. The condition of grave general intoxication is manifest primarily in functional disturbances of the central nervous system. Also of major significance is the septic affection of the visceral organs: the heart, kidneys, liver, intestine, etc. The patient's general condition is greatly impaired, his pulse is very quick and thready, and hemorrhages begin to occur into his visceral organs, under the skin and mucous membranes. The skin often becomes yellow.

Treatment. A thorough examination of the ear, nose and throat should be made in case of septicemia of unknown etiology. The diagnosis having been made, an immediate operation is necessary; in acute suppurative otitis simple trephination is sufficient, while in the chronic form a radical operation is required. When the affected sinus has been laid open, its wall is incised to remove the clot. The affected bone should be removed until healthy bone is encountered. Postoperative treatment consists in careful attendance on the patient, skin care is essential, his teeth and tongue should be cleansed, his stool must be regular, etc. It is important to observe the activity of the heart and to institute penicillin treatment every three hours in 100,000 unit doses for a period of 10 to 12 days. A combined use of penicillin and streptomycin is even more effective. The patient's diet

should be sufficient in quantity and rich in vitamins. Timely surgical interference, blood transfusion and antibiotic treatment have largely improved the chances of recovery from this grave disease. Today, recovery is certain in nearly all cases.

Cerebral Abscess

(*Abscessus cerebri*)

Otitic cerebral abscess is usually located in proximity to the primary focus of infection in the middle ear. A lesion of the *tegmen tympani* and antrum in most cases causes an abscess in the temporal lobe.

An abscess in the cerebellum may occur by invasion of the infection through the internal auditory meatus following labyrinthine suppuration or from a simultaneous lateral sinus thrombosis.

Symptoms. At first, the signs of a cerebral abscess are quite unmarked and, therefore, are not infrequently ascribed to other diseases. The typical symptoms are stubborn headache at normal body temperature, recurrent attacks of vomiting and a slow pulse rate. Lethargy, inertness and slow reaction are also frequent signs. The patient answers questions with reluctance and after an interval becomes drowsy and indifferent to his surroundings. An abscess in the left temporal bone often produces amnestic aphasia, in which the patient cannot recall the names of common objects, though he can explain their use. In half of the cases there is a change in the eye fundus, the choking of the optic disc.

Diagnosis. Diagnosis of cerebral abscesses often presents difficulties. The most typical cerebral symptoms of general character—headache, vomiting and slow pulse rate—may be lacking at the initial stage and become pronounced only by the end of the disease, which makes prognosis difficult. An abscess in the cerebellum brings about cerebellar ataxia and nystagmus which tends to increase in the eyes turned towards the affected side.

Treatment. Cerebral abscesses are treated surgically. Following a simple or radical operation on the middle ear and removal of the primary focus of infection, an extensive exposure of the dura mater should be made. A search for the exact location of the abscess by tentative punctures of the

brain should be primarily confined to the areas of pathological changes in the dura mater. After the abscess has been located, it should be opened after a preliminary incision of the dura mater. The subsequent treatment is drainage of the abscess, daily change of dry dressings and administration of penicillin or streptomycin, separately or together. Another method is repeated punctures followed by aspiration of pus and antibiotic douches of the abscess.

The patient should be given absolute quiet and careful nursing. Recovery is observed in six to eight cases out of ten. In abscesses of the cerebellum the recovery rate is less.

Purulent Meningitis

(*Meningitis purulenta*)

Until recently suppurative inflammation of the pia mater was a fatal disease.

Symptoms. Purulent meningitis begins with intense headache and a high temperature. Prodromal symptoms, such as a slight unilateral headache, fits of vertigo, an occasional low fever, are often observed for a period of a few days. Nausea and vomiting frequently accompany the onset of the disease, particularly in children. The pulse rate is fast and irregular. The most typical features of meningitis are symptoms of irritation of the meninges, such as stiff neck, i.e., rigidity of the neck muscles which prevents flexion of the chin on the chest, and positive Kernig's sign, i.e., the leg flexed at right angles at the thigh joint cannot be straightened out at the knee. The most valuable guide in diagnosing meningitis is examination of the cerebrospinal fluid obtained by lumbar puncture, which should be performed by a physician. In meningitis the fluid may be turbid, containing numerous cells and sometimes bacteria, and flows out under pressure. The development of meningitis is accompanied by progressive stupor and quick aggravation of the patient's general condition together with respiratory disorders, incontinence of urine and feces. The duration of the disease varies from one to two days in the so-called lightning forms and seven to ten days in other cases.

Treatment. A simple or radical operation should be immediately undertaken to remove the primary focus of infec-

tion in the middle ear. This should be followed by intensive chemotherapy with large sulfonamide doses of up to 10 g in 24 hours, or penicillin injected intramuscularly in 100,000 unit doses every two or three hours to make a daily dosage of up to a million units. Still better results have been given by these drugs in combination, as well as by combination of penicillin with streptomycin. The daily dosage of sulfonamide should be up to 8-10 g. Intracranial pressure is relieved, if required, by repeated lumbar punctures to let out part of the cerebrospinal fluid. In a similar manner penicillin in a 50,000 unit dose may be infused into the spine to aid recovery.

Nursing a patient with an intracranial complication is a matter of major importance, because this condition is often an extremely grave one and requires special attendance. This combined with a timely and accurate fulfilment of the doctor's instructions by an efficient nurse will ensure success of the treatment and save the patient's life.

NONSUPPURATIVE DISEASES OF THE MIDDLE AND INTERNAL EAR

Chronic Catarrh of the Middle Ear

Chronic catarrhal otitis media is produced by various morbid processes in the nose and nasopharynx which spread up the Eustachian tube and serve to narrow its lumen thereby obstructing ventilation of the middle ear. Repeated acute catarrhs of the middle ear gradually thicken its mucosa and make the drum less elastic. A long-standing obstruction of the Eustachian tube gradually leads to a noticeable and stubborn retraction of the drum followed by ankylosis of the ossicular chain. Frequently lengthwise and crosswise fibres of scar tissue form between the drum and the walls of the tympanic cavity. This condition is known as chronic or adhesive catarrh. The patient complains of progressive deafness and tinnitus. It often happens that the hearing improves at times, particularly in dry weather, and deteriorates when the weather is damp, and in coryza.

Diagnosis. The diagnosis of this condition rests on examination of the drum and functional examination of hear-

ing. The drum is more or less markedly indrawn (Fig. 31), dull and sometimes creamy-white in colour. Sharply outlined white spots of variable form are often observed, which are calcareous deposits in the depth of drum tissue. Scars left by suppurative otitis, as well as atrophic areas of the drum appear to be dark and are often mistaken for drum perforations. In atrophy the drum closely adheres to the internal wall of the middle ear, which sometimes creates the impression of complete absence of the drum.

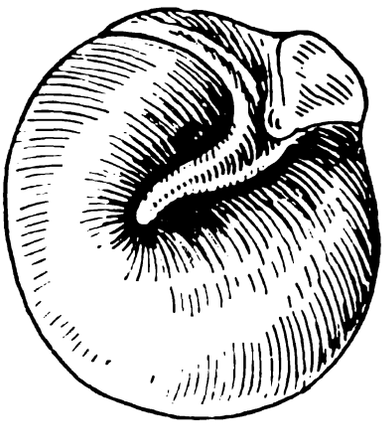


Fig. 31. Drum Retraction

The extent of changes in the drum has no decisive bearing on the degree of hearing. Therefore, the diagnosis should be verified by an assessment of the hearing and in many cases by a trial inflation of the auditory tube. The most typical results will be produced by tuning-fork tests where a nearly normal hearing for high tones produced by a C_{2048} tuning fork is accompanied by a severe low-tone loss as evidenced by the use of a C_{128} tuning fork. Bone conduction is often lengthened.

Trial inflation of the tube often improves the hearing immediately.

Prognosis. This is favourable if the disease is of short duration, and the hearing has markedly improved after tubal inflation.

Treatment. The first task is to restore the patency of the Eustachian tube, that is, to eliminate the morbid condition in the nose and nasopharynx. Adenoidectomy is a frequent procedure in such cases, particularly in children, while operations on adults are mostly performed for deformities of the nasal septum, for removal of hypertrophic posterior ends of the lower nasal conchae, etc.

Sometimes, these measures alone are sufficient to remove the tubal obstruction and largely restore the hearing. But should elimination of the nasal disease fail to produce a lasting improvement of hearing, tubal inflation will be required.

Tubal inflation is carried out by means of a rubber bulb

Fig. 32. Rubber Bulb for Tubal Inflation

(Fig. 32) through an olive-shaped composition tip or an aural catheter. The first procedure is based on the fact that in swallowing and pronouncing some consonants and vowels the soft palate rises and fully closes the entrance to the nasopharynx. By pressing on the bulb at this moment the air in the nasal cavities will be compressed and pushed into both Eustachian tubes. Inflation is performed by introducing a composition tip into one of the nostrils which are pinched silmultaneously with fingers of the left hand. The patient is directed to take a little water into his mouth and swallow it at the count of three. At this moment the bulb is compressed, and a blast of air penetrates into the Eustachian tubes with a characteristic noise.

Tubal inflation may also be performed without the use of water. The patient is directed to call out numbers, and at the count of three an air blast from the bulb is blown into the ear. To check whether inflation has been successful an otoscope is used (Figs. 33a and 33b). This is a slender rubber tube with composition ear tips at either end for insertion into the ear to be inflated and the examiner's ear, as shown in Fig. 33b.

In cases where a rubber bulb and composition tip are inadequate equipment for inflation to be properly performed, or if unilateral inflation has to be made, the tube will be inflated through a catheter following a short nasal anesthesia, if necessary. An aural catheter (Fig. 34) is a slender 15 to 17 cm long metal tube curved like a beak

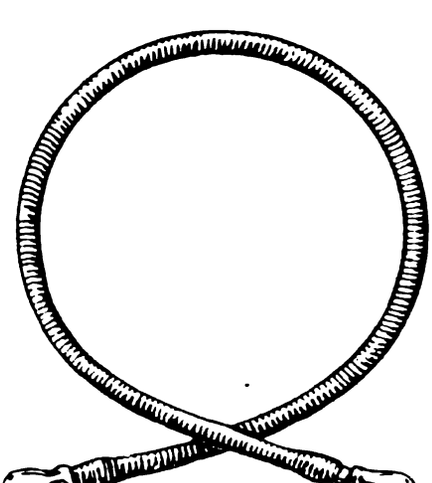


Fig. 33a. Otoscope

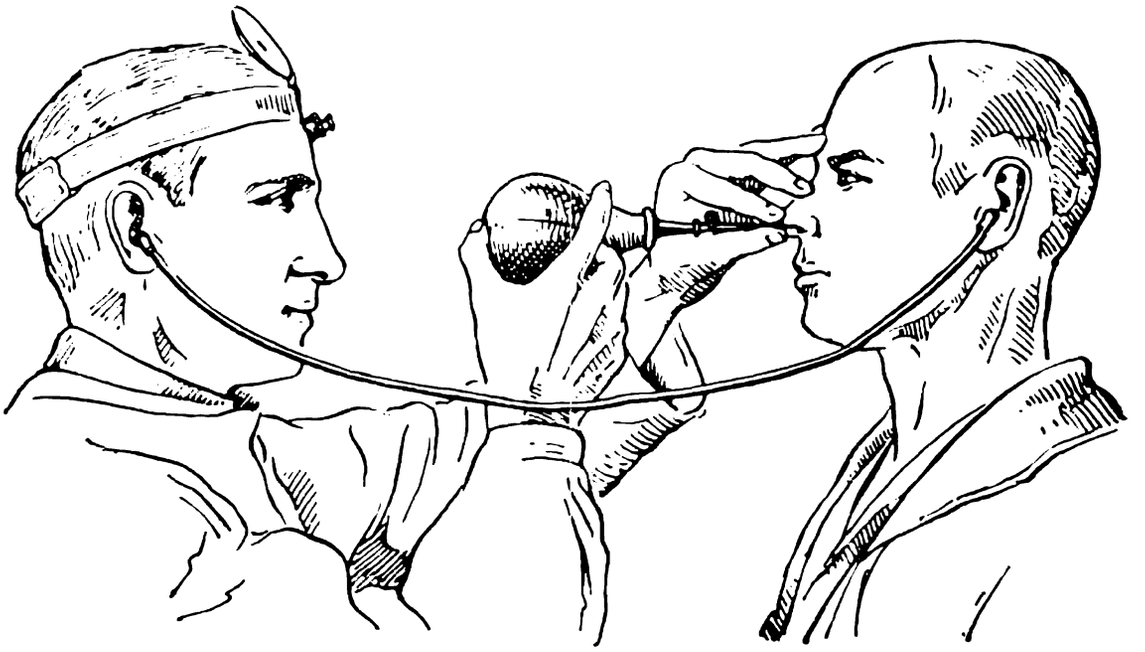


Fig. 33b. Otoscope Inserted into Examiner's and Patient's Ears

at one end and funnel-shaped at the other, basal end. At the base of the catheter, there is a small ring set on edge in the same plane as the beak. Prior to use, the catheter should be sterilised in boiling water. After the catheter has been slipped in along the nasal floor down to the nasopharynx with its beak pointing downwards, the latter is turned to the middle, and the catheter is gently pulled back until the beak has touched the back edge of the vomer. Here, on the lateral wall of the nasopharynx, is the mouth of the Eustachian tube. By turning the beak 180 degrees outwards it is slipped into the mouth of the Eustachian tube. This is followed by inflation (Fig. 35). The catheter should be introduced with gentle caution and without any pressure. The beak curvature may be altered, if necessary.

When air is blown through the catheter, characteristic sounds may be heard through the otoscope. These may vary

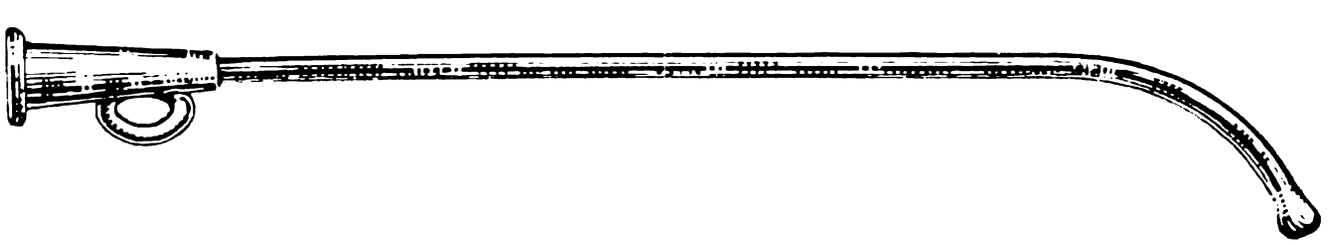


Fig. 34. Aural Catheter

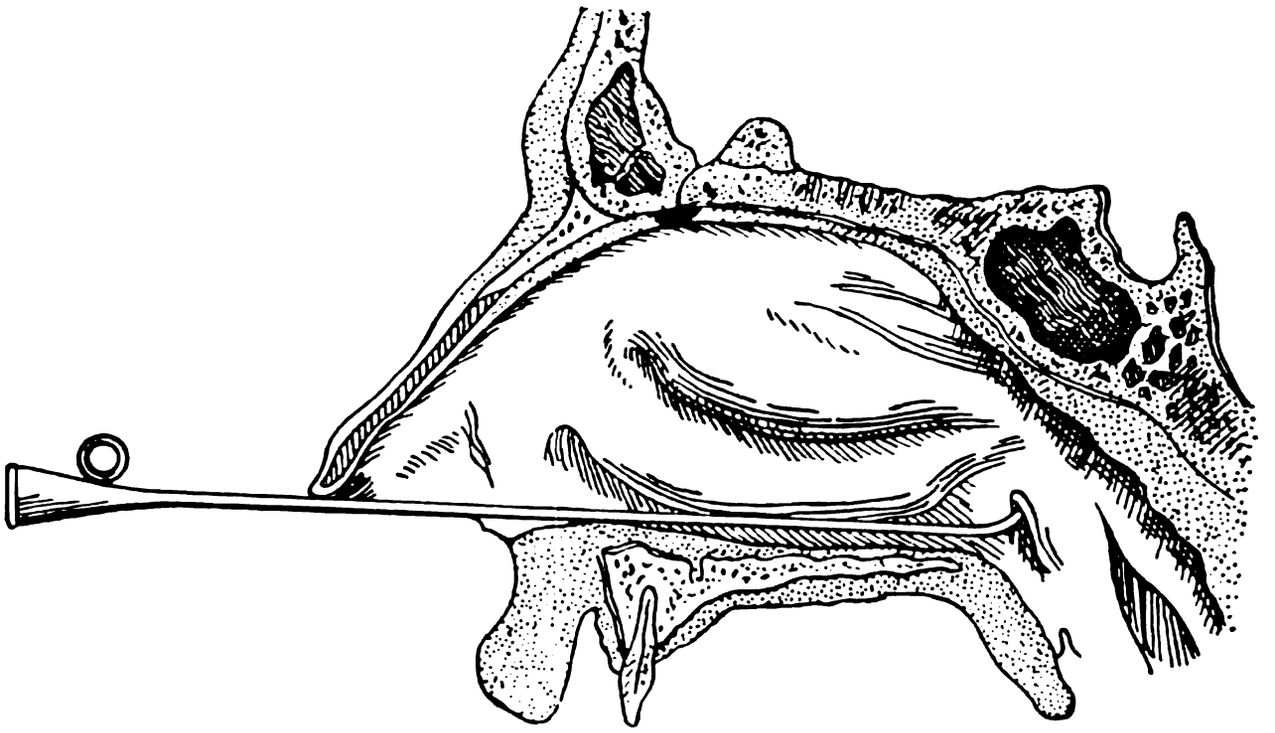


Fig. 35. **Tubal Inflation through Catheter**

according to the patency of the Eustachian tube and its possible mucous contents. A soft blowing sound indicates a patent tube, louder high-pitched sounds are a sign of tubal obstruction, and, finally, the presence of exudate causes characteristic bubbling sounds. Careless insertion of the catheter may injure the mucous membrane and produce nasal bleeding. The blowing of air into torn tissues may cause emphysema.

In severe atrophy of the drum, inflation should be made with great care and sometimes be abandoned for fear of rupturing the drum.

Inflation may improve the hearing for several hours to a few days. Therefore, repeated inflations have to be made every one, two or three days, sometimes up to 5, 10 and 15 times in all. The nasopharynx is simultaneously painted with 1 % silver nitrate solution or 0.25 % Lugol's solution. In advanced cases, various kinds of thermic procedures, diathermy and mudtherapy are used to resolve commissures and increase flexibility of the ossicles, which unquestionably aid recovery. A pneumatic massage of the drum can also be used in combination with inflation. If a special apparatus is not available, the massage can be made by

means of a pneumatic speculum tightly pressed into the auditory canal and compressed with moderate effort up to 60-100 times a minute to produce alternate suction and pressure on the drum membrane. This will make the drum move in and out and set in motion the entire ossicular chain. In recent time, injections of aloe preparations and skin grafting by Filatov's method have been used with favourable results.

Prophylaxis. The best way to avert middle ear catarrh is to ensure normal nasal breathing. The earliest possible treatment of acute catarrh of the upper respiratory tract and timely management of chronic diseases of the nose and nasopharynx will no doubt serve to keep down the rate of severe deafness.

Prophylaxis of amblyacousia (dull hearing) in pre-school and school children demands the utmost attention. Periodical examination of all children of this age always reveals those who are in need of some treatment. The presence of adenoids severely affects the hearing and their timely removal will undoubtedly prevent hearing loss in quite a number of cases.

Otosclerosis

Otosclerosis is a peculiar disease of the bone capsule of the labyrinth commonly found in young people, and characterised by progressive deafness and tinnitus.

Etiology. The cause of the condition is unknown. It occurs most commonly in females aged between 15 and 25; pregnancy and labour have an adverse effect on the hearing.

Pathological changes in otosclerosis consist in a partial conversion of the hard bone around the labyrinth into a spongy osteoid tissue with widened marrow spaces and Haversian canals containing loose connective tissue with a ramified vascular network. The spongy bone is commonly laid down around the oval window and on the promontory of the internal wall of the middle ear, then on the cochlear apex and around the circumference of the internal auditory meatus. The morbid process which has originated at the

oval window invades the stapes and in time brings about its complete ankylosis and immurement in the oval window.

The patient complains of a progressive loss of hearing and tinnitus which may often be most oppressive. Physical examination will reveal a normal drum and auditory tube. At first the deafness is of the conduction type due to ankylosis of the stapes until the disease has advanced so far as to invade the cochlea and in addition cause nerve deafness. Paradoxical hearing (*paracusis Willisii*) is frequently to be observed in otosclerosis where the patient hears best in noisy surroundings. Some believe that it is due to loud sounds producing concussion of the stapes which is only beginning to ossify. A differential diagnosis of otosclerosis is sometimes rather difficult, as it rests between chronic catarrh of the middle ear and neuritis of the acoustic nerve.

Course. Progressive impairment of hearing sometimes has brief periods of remission; in most cases, however, an unfavourable factor of some kind, such as labour, infectious diseases, mental shock, etc., is followed by a rapid deterioration of the condition. The process is commonly bilateral, though sometimes hearing loss in one ear is more rapid than in the other.

Treatment. The etiology of otosclerosis is still unknown. Consequently general prophylaxis in the treatment of this disease is of particular importance. The patient himself notes a certain hearing loss during hard manual work and in nervous strain. These facts should be borne in mind in private life and at work. In some cases, the treatment of chronic catarrhal otitis present in otosclerosis serves to improve the patient's condition and, should the results of treatment be negative, helps to verify the diagnosis of otosclerosis. Recommended remedies are iodine, phosphorus, phytin and bromine drugs to be taken in fractional doses for a long period. In some instances their use will arrest the rapid progress of the disease and may partially restore the hearing.

Hearing exercises and lip reading often prove successful in the treatment of otosclerosis as well as in some forms of neuritis.

Bromine drugs in combination with luminal decrease the heightened excitability of the central nervous system there-

by lessening tinnitus which has a negative effect on the patient's general condition.

Recently, otosclerosis has been treated surgically. The operation, fenestration, consists in making an artificial opening or window (*fenestra*) in the external bony semicircular canal for the passage of sound waves into the labyrinth.

Apart from fenestration, Rosen's stapes mobilisation is often used to restore the mobility of the stapes ankylosed as a result of otosclerosis. If this method fails, one or a few openings are made in the foot plate of the stapes, which is known as fenestration of the foot plate of the stapes. The latter may also be completely removed.

Neuritis of the Acoustic Nerve

(*Neuritis nervi acustici*)

Diseases producing acoustic nerve degeneration and atrophy have been classified under the general name of neuritis of the acoustic nerve.

This condition may be caused by intoxication, infection, metabolic and circulatory disturbances.

Degeneration and atrophy of the nerve endings and the acoustic nerve trunk produce a gradual and lasting loss of hearing characterised by a lowered high-tone limit and a drastic shortening of the high-tone range (tuning fork C^4); a considerable shortening of bone conduction is to be noted at the same time.

The hearing for conversational speech may remain more or less satisfactory for a long time.

1. **Toxic neuritis.** Severe forms of this kind of neuritis may be caused by excessive administration of quinine, salicylic acid, streptomycin, mercurial preparations, arsenic and some other drugs.

It should be borne in mind that excessive dosage and prolonged use of streptomycin may lead to a large or total loss of hearing in hypersensitive subjects. Yet the vestibular department of the labyrinth more often succumbs to subsequent labyrinthine disturbances.

Chronic intoxication may result from addiction to alcohol and tobacco, as well as from a protracted effect of various

industrial chemicals. The lesion is usually bilateral and primarily envelops the peripheral endings of the acoustic nerve, and later but more rarely extends to the vestibular nerve.

In severe intoxication, prognosis is more hopeful, as there is a chance of full restoration of hearing, whereas in chronic cases the advanced degeneration of the middle ear does not respond to treatment.

2. **Infectious neuritis.** Nearly all infectious diseases may entail an affection of the internal ear, but the highest rate of such complications is observed in cerebrospinal meningitis, scarlet fever, mumps, measles, influenza, and typhoids. Diseases of the internal ear also occur in congential and acquired syphilis. Specific changes peculiar to a particular kind of infection are produced in the cochlea and the acoustic nerve trunk by the entry of bacteria or their toxins into the labyrinth. In such diseases as, for instance, spotted and relapsing fever (other infectious diseases should not be excluded) changes are noted not only in the peripheral nerve endings of the auditory analysor but also in the peripheral nerves, the acoustic nerve nuclei, the medulla oblongata, and the central cortical sections of the auditory analysor (organ of Corti).

Owing to the tremendous capacity for functional compensation of some sections of the cerebral cortex by others, repeatedly proved by I. P. Pavlov, cortical disturbances of hearing often disappear without leaving a trace, whereas affections of the peripheral nerve endings in the cochlea are persistent phenomena.

Treatment. The treatment of postinfectious neuritis of the acoustic nerve is by employment of a hygienic regimen following recovery from an infectious disease and by the use of various methods of physical medicine to improve the patient's general state of health by building up his body resistance. Chemotherapy is with pilocarpine, fractional doses of iodine, strychnine, proserinum, dibasolum and endocrinic drugs, as well as vitamin B_1 and nicotinic acid. The earlier the commencement of treatment, the more hope there is for improvement in the hearing. Moreover, in early cases of infectious neuritis, intramuscular penicillin injections are advisable. Tissue therapy is also effective to a certain extent.

In other cases, considerable changes in the internal ear are caused either by metabolic disturbances which occur in a number of diseases, diabetes in particular, or by the effect of blood toxins, as in nephritis or gout.

The most common cause of neuritis is a disturbed supply of the internal ear in atherosclerosis and vascular hypertension requiring general treatment of the basic disease.

DEAF-MUTISM

Deaf-mutism is due to total bilateral deafness, either congenital or acquired in early childhood, approximately before three years of age. The child who cannot hear spoken speech is unable to learn to speak or forgets the speech it had learned before. In other words, mutism is the result of deafness. The cause of congenital deafness is maldevelopment of the labyrinth or labyrinthine diseases during fetal life. Congenital deafness is ascribed by some scientists abroad to hereditary factors alone ignoring the effects of unfavourable socio-economic conditions. Meanwhile, it has been noted that in the U.S.S.R. the decline in the number of deaf-mute children by two-thirds in the past three decades coincided with the advancement of the material and cultural standards of the people.

Acquired deafness occurs more often than congenital, and often results from an infectious disease. The chief cause is cerebrospinal meningitis; the second, scarlet fever which affects the middle and internal ears, and also measles. Less frequently deafness is due to typhoids, syphilis, diphtheria, mumps, influenza and whooping cough.

Among other important causes of deafness is injury to the skull. Such factors as jamming of the fetal head in a narrow pelvis, application of forceps or prolonged asphyxia may eventually lead to deafness.

In congenital deafness, the vestibular function is often unaffected, whereas in acquired deafness both labyrinthine functions are nearly always affected owing to the spread of the inflammatory process within the inner ear or in the nerve trunk.

The deaf-mute can utter sounds and may be trained to talk. Since he cannot hear, his voice is monotonous. The

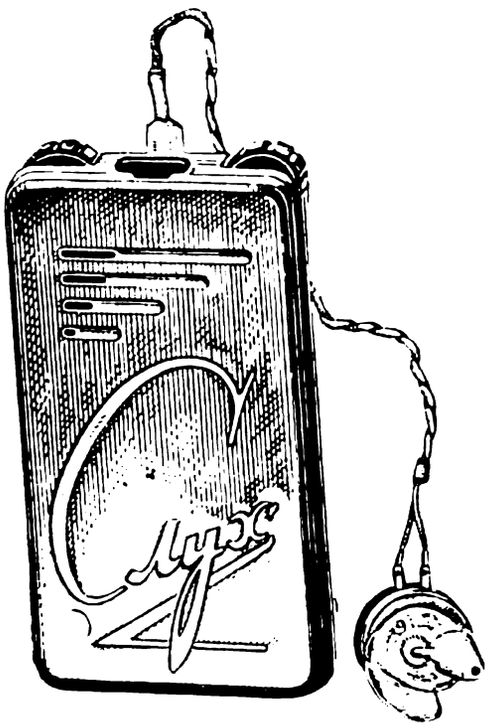


Fig. 36. Hearing Aid with Air and Bone Conduction Telephone Receiver

diagnosis of deaf-mutism in infancy is a very complicated task. Factors to be noted are the parents' evidence of absence of reaction to sounds by the child, as well as the results of whistling and tuning-fork tests.

A number of unconditioned reflexes are used to determine the presence of hearing, such as the auropalpebral reflex, or the reflex winking of the eyes, and the auropupillary reflex, i.e., the narrowing and widening of the pupils when a sounding tuning fork is held near a hearing ear. In more difficult cases, experiments with conditioned reflexes have to be used to determine the presence of hearing.

Treatment. The treatment of deaf-mutism has so far met with little success. Therefore, the basic approach here is to give the deaf-mute speech training by making use of vision, the sense of touch and residual hearing, if any exists. This training is carried out in special schools and kindergartens. The methods of instruction are selected in relation to age, the degree of residual hearing and other individual features. Many deaf-mutes have not only learned to talk and studied the elements of science, but they have graduated from higher schools. There are deaf-mute scientists, artists and outstanding specialists in various fields.

Hearing aids. Recovery from an ear disease may be accompanied by more or less advanced permanent deafness handicapping the patient in his intercourse with other people and in certain useful activities. In such cases, electrical aids to hearing are recommended, similar to that shown in Fig. 36. Today, two types of hearing aids are manufactured in the Soviet Union: (a) a portable microtelephonic hearing aid fed on a torch battery, and (b) a radio-valve hearing aid of much greater amplifying capacity.

Recently, transistor hearing aids built in a spectacles frame have been manufactured. Hearing aids are selected at special aural rehabilitation centres.

TRAUMATIC LESIONS OF THE EAR

Trauma of the External Ear

The unprotected position of the auricle makes it liable to frequent injuries in everyday life and in industry. Contusions or mechanical injuries produce lacerations in the aural skin or cartilage with formation of othematoma, perichondritis or even a phlegmon.

Othematoma. Bruising or continuous pressure often produces a collection of serous and serosanguineous fluid resembling a bluish-red swelling of a rubbery or fluctuating feel located mostly in the upper half of the pinna without any great tenderness in the surrounding tissue. Small othematomas are treated with cold, wet dressings, pressure bandages and paintings with 5% iodine tincture. The treatment of a large othematoma is by puncture and pus aspiration with strict observance of aseptic rules, followed by application of a pressure bandage. In the case of an obvious infection, free incision is indicated with antibiotic treatment, as well as "sollux" and ultraviolet irradiation.

The most serious sequel of all kinds of aural trauma with skin laceration is inflammation of the perichondrium, that is auricular perichondritis. The auricle becomes markedly swollen, red and very tender. A long process of this kind will culminate in necrosis of the cartilage and shrinking of the auricle, which turns into a shapeless lump of scarred tissue.

Treatment. The traumatic wound should be immediately sutured and protected with an aseptic dressing. If stitching is performed shortly after the accident, healing will be rapid, and even the torn-off pieces of the auricle will heal. In case of suppuration and necrosis of cartilage, treatment is by incision and scraping off the dead cartilage, as well as by intramuscular injections of penicillin and streptomycin. Injuries of the auricle and the membrano-cartilaginous part of the auditory canal are treated by plastic surgery and tamponade to prevent atresia of the auditory meatus.

Isolated injuries of the bony portion of the external auditory meatus may occur in fractures of the mandibular joint. Gunshot wounds may involve severe combined traumas of the ear with a simultaneous injury to the external auditory meatus, the mastoid process, the tympanic cavity or the joint of the lower jaw. As a rule, such wounds cause rupture of the drum and later are frequently followed by suppurative otitis media.

The treatment of these wounds consists in their primary surgical management, removal of loose fragments, provision for free drainage through the external auditory meatus, as well as in general and local chemotherapy with antibiotics and sulfonamides.

Traumatic Rupture of the Tympanic Membrane

The drum may be ruptured in various ways: by a flat-handed slap on the ear, in diving and even by a kiss on the ear. In the first two cases, the cause of rupture is a sudden compression of the air in the auditory canal, while in the last instance, the suction and rarefaction of air may have the same outcome.

The drum may be perforated by thin tree branches, dry stalks of plants, knitting needles and crochets inserted into the auditory canal.

Another cause of injury to the drum is inexpert manipulation of an unsuitable instrument, such as an aural pincers, to remove a foreign body from the ear.

As a rule, the danger lies not so much in the drum rupture itself as in acute suppurative otitis media, which often occurs owing to the entrance of infectious germs through the perforation.

Prior to the onset of otitis media, the drum rupture is identified by a slit-like or torn perforation of irregular form with blood visible around its edges, as well as by the absence in the drum of obvious inflammatory symptoms.

In war-time, drum rupture may be the result of direct, mostly gunshot injuries of the middle and external ears, as well as of the impact on the ear of air blasts from shell, bomb and mine explosions.

The treatment of isolated drum ruptures is by cleansing the outer portion of the auditory canal of dried and congested blood with dry sterile cotton wool. Ear douches and even drop instillation of any kind should be avoided as a precaution against middle ear infection.

Injuries of the Middle and Internal Ear

Cases of isolated injuries to the middle and internal ears are rare. They primarily refer to gunshot wounds which commonly involve a simultaneous injury to the adjacent tissues, for the most part penetrating wounds of the skull. A direct lesion of the middle ear affects the mastoid process, and a fracture of the facial nerve canal leads to facial nerve paralysis. Wounds in the aural region often involve the mandibular joint. Injuries in and around the middle cranial fossa are often accompanied by fissures in the roof of the middle ear and ruptures of the drum. These injuries cause an outflow of cerebrospinal fluid from the ear.

An injury of the internal ear is followed by a series of disturbances in hearing and equilibrium. The common signs are tinnitus, impaired hearing, giddiness, nausea, and even vomiting. Moreover, the patient is unable to walk and even stand erect. Nystagmus is an almost constant symptom during this stage.

The patient should be strictly confined to bed and given large doses of antibiotics and sulfonamides.

Continuous bleeding or suppuration may necessitate surgical interference.

In less severe cases, the treatment of residual otitis should be conducted by ordinary means. It should be remembered, however, that otitis following a gunshot wound is very likely to produce a complication and demand surgical interference.

Air Concussion of the Ear

Noise and concussion from bomb, mine and shell explosions cause a number of disturbances in the body, such as substantial functional and anatomical changes in the ear. The impact of a blast is usually combined with tremendous irritation by sound. These factors simultaneously con-

tribute to an affection of both the peripheral nerve endings of the auditory analysor and the central nervous system. The most conspicuous signs are injuries of the central nervous system, such as contusion or concussion of the brain, which are indicated by stupor, mental depression, total deafness or loss of speech and hearing, as well as a number of other neurological symptoms common to the shellshocked.

Deafness often follows the impact of a blast on the middle and internal ears. The most usual symptoms of this condition are rupture or complete disorganisation of the drum membrane, hemorrhage and anatomical displacement in the cochlea and semicircular canals.

The shellshocked commonly complain of tinnitus, feebleness, vertigo, and headache. Later, degeneration of the peripheral nerve endings of the auditory nerve may follow together with a considerable loss of hearing.

Treatment. In curing the shellshocked attention should first be given to the state of their central nervous system and mentality, because concussion deafness is largely caused by an extensive inhibition of the cerebral cortex known as transliminal inhibition, which is the result of overstimulation of the brain. Therefore, protective inhibition, sleep therapy, is the most effective in appropriate cases.

Total concussion deafness, sometimes combined with total speech loss, persists for a few days to several weeks. Sometimes, these symptoms suddenly disappear under the influence of strong emotional experience. More commonly, however, restoration of hearing is a gradual process. Speech returns sooner than hearing and, as a rule, immediately. Tinnitus and deafness are treated in the same way as neuritis of the acoustic nerve. In stubborn cases of severe deafness a hearing aid may be recommended.

OCCUPATIONAL DISEASES OF THE EAR

The causes of an occupational ear disease may be the following: (1) continuous shrill sound, noise and vibration; (2) high or low atmospheric pressure; (3) industrial poisoning.

1. Among the so-called noisy occupations are the trades of boiler-maker, riveter, motor-tester, weaver, nail worker,

etc. Protracted noise and vibration give rise to deafness, whose degree depends on the length of time worked, the nature and intensity of noise.

Experiment has proved that deafness is produced by degeneration of the organ of Corti and the trunk of the acoustic nerve. In noisy industries, the effect of noise transmitted through the air or bone is often aggravated by the tremulous effect of low-frequency vibrations.

2. The effects of high and low atmospheric pressure are to be noted in coffer-dam workers, deep-sea divers and aviators. A patent auditory tube maintains a balance between the changing atmospheric pressure and air pressure inside the middle ear when a plane lands or takes off, or when air is pumped in or out of a coffer dam. Partial or total obstruction of the Eustachian tube will interfere with this process and cause extraordinarily painful retraction or bulging of the drum and possibly its rupture. A quick drop in middle ear pressure may lead to hyperemia *ex vacuo* with dilation of blood vessels and transudation; rupture of individual vessels followed by hemorrhage is also likely to happen.

A quick fall of high atmospheric pressure, for instance, in case of a too early exit from a coffer dam, can lead to aeroembolism, commonly known as the bends or caisson disease.

A prolonged exposure to increased atmospheric pressure will result in excessive nitrogen being accumulated in the tissues of the body which at a sudden drop in pressure, as when leaving a coffer dam is not expelled from the lungs together with the air exhaled. In such circumstances, the nitrogen bubbles circulating in the blood may block some of the vital vessels, as well as the vessels of the internal ear. A sudden rise of atmospheric pressure in a caisson may cause severe pain and rupture of the drum membrane.

3. Among industrial chemicals, those most harmful for the inner ear are lead and lead compounds. Isolated cases of ear affection have been noted in regular work with mercury, arsenic and benzol, as well as in carbon monoxide poisoning.

The number of trades involving lead hazards in the U.S.S.R. has been reduced to the minimum by using harm-

less alloys instead of lead. Cases of lead poisoning are now exceedingly rare.

Owing to a long series of measures, occupational diseases, amblyacousia in particular, in workers exposed to noise hazards are seldom met with in the U.S.S.R., whereas in pre-revolutionary Russia their incidence was fairly high, as shown by the statistical data available. The concern of the Communist Party of the Soviet Union and the Government for the workers' living and cultural standards, as well as a shorter working day, paid holidays, labour safety rules and sanitary regulations for industrial plants have, naturally, favourably affected the health of the workers. This has made occupational diseases a very rare occurrence even in noisy industries. For their part, the welfare organisations take care that the required prophylactic measures are implemented without delay.

Prophylaxis. Social and personal hygiene is indicated. Individual prophylaxis against industrial noise is less effective, since stopping the ears with cotton wool, the use of various sound-proof contrivances and cork helmets are all useless against bone conduction.

Collective prophylaxis consists in technical improvements and removal of the causes of harmful effects. Thus, elimination of industrial noise may be achieved by the substitution of one method of metal-working for another. For instance, boiler riveting and metal cutting which had formerly been extremely noisy operations have been largely replaced by noiseless electric welding and cutting. Welding is now used in building huge metal structures: skyscrapers, bridges, ships, steam-boilers, etc. Substitution of plastics for metal parts is also a step in the same direction.

Noise can be lessened by careful maintenance of machine-tools and timely correction of all defects producing additional noise.

Plans of industrial projects must include sanitary regulations governing noise and vibration limits.

In prevention of caisson ear disease, there should be strict observance of the existing rules and instructions for work at a high atmospheric pressure. This refers above all to selection of the right length of time to pump air in or out of a coffer dam.

Newly-employed workers undergo medical examination to determine their fitness for a particular job or for a branch of industry listed in appropriate legislation.

People with ear diseases should not be employed on work harmful to hearing, such as in noisy industries and in coffer dams.

People wishing to be employed on work where normal hearing is necessary, such as lorry drivers, members of train crews, telephone and telegraph operators, should also undergo hearing tests. Anyone unable to hear whispered speech at a distance of less than six metres, even if this phenomenon is unilateral, is unfit for any of the above-mentioned jobs.

DISEASES OF THE NOSE, PHARYNX AND LARYNX

DISEASES OF THE NOSE AND PARANASAL SINUSES

ANATOMY OF THE NOSE

The nose is the frontal department of the upper respiratory tract (Fig. 37), and is subdivided into the external nose and the nasal cavity with the paranasal, or nasal accessory, sinuses. The external nose is a triangular pyramid of irregular shape composed of bone, cartilage and soft tissue. The upper angle of the pyramid adjoining the forehead is the root of the nose which extends downwards and outwards to form the nasal dorsum rounded with the tip of the nose at the lower free angle. The lateral flaring and flexible expansions of the nose are known as the *ala nasi* or wings of the nose, whose free lower margins are so shaped as to form the nostrils or anterior nares.

The Nasal Cavity

The nasal cavity borders on the cranial cavity above, the eye sockets on the sides, and the oral cavity below. The nasal septum divides the nasal cavity into two halves, which open out through the nostrils facing downwards at a slightly oblique angle. Posteriorly, the nasal cavity communicates with the nasopharynx through two neighbouring orifices of oval shape called the choanae.

The posterosuperior bony part of the nasal septum is made up of the vomer and the perpendicular plate of the ethmoid bone, while the anteroinferior cartilaginous part is formed by the quadrangular cartilage.

The external or lateral wall of the nasal cavity (Fig. 38) is the most complex structure, as well as the most important region from the practical aspect. Starting from the external nose, it is made up of the following bones: the

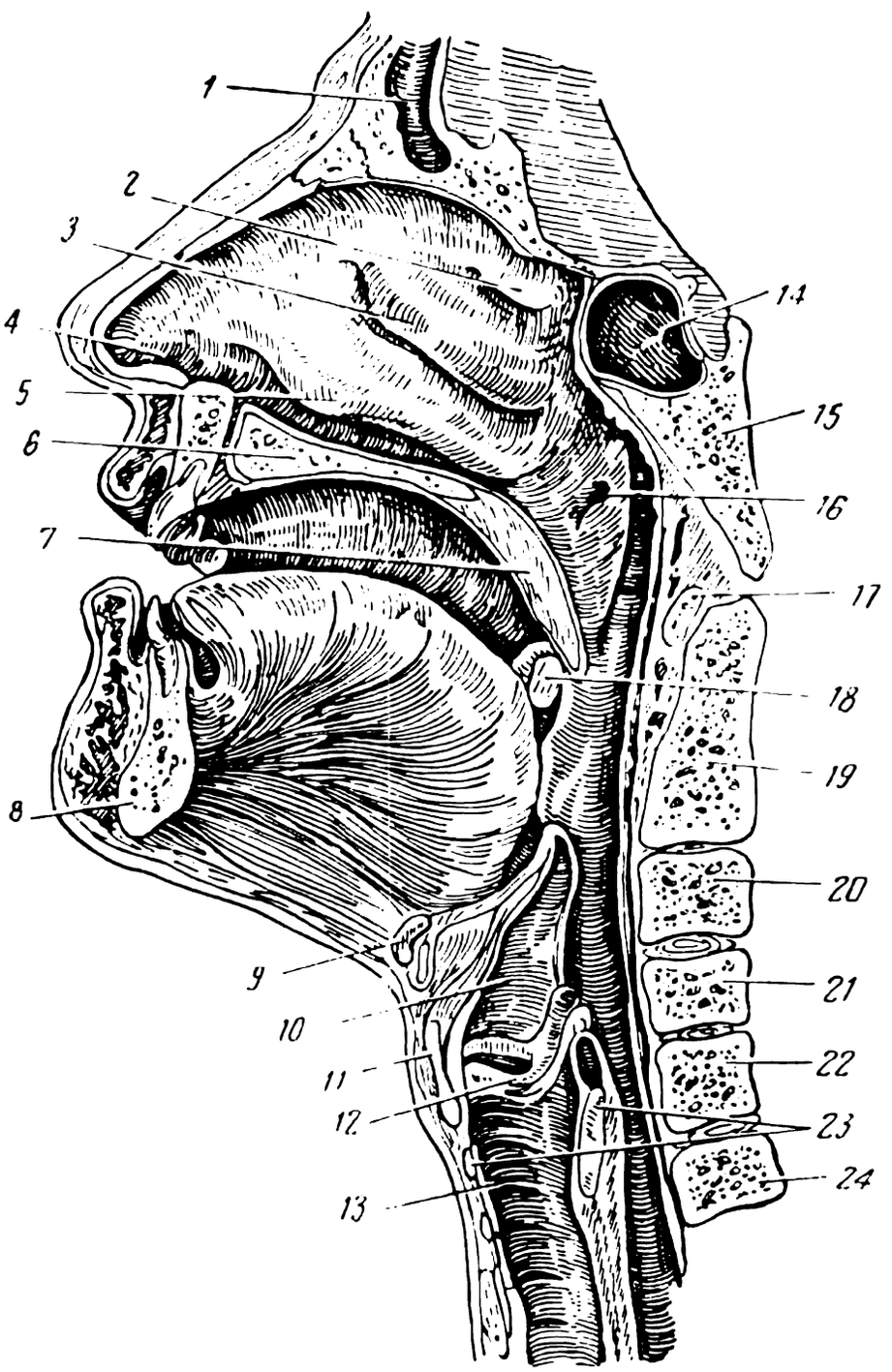


Fig. 37. Saggital Section of the Nasal Cavity, Pharynx and Larynx

(1) frontal sinus; (2) superior turbinate; (3) middle turbinate; (4) nasal vestibule; (5) inferior turbinate; (6) hard palate; (7) soft palate; (8) mandible; (9) hyoid (lingual) bone; (10) epiglottis; (11) thyroid cartilage; (12) true vocal cord; (13) trachea; (14) sphenoid sinus; (15) body of sphenoid bone; (16) pharyngeal opening of auditory tube; (17) arch of atlas; (18) palatine tonsil; (19), (20), (21), (22), (24) 2nd, 3rd, 4th, 5th, 6th cervical vertebrae; (23) cricoid cartilage

nasal bone, the frontal process and the nasal side of the maxilla, the lacrimal bone, the ethmoid bone, the palatine bone and the alar processes of the sphenoid bone.

Three ridges known as the conchae or turbinates spring from the *lateral wall* of the nasal cavity to divide it into

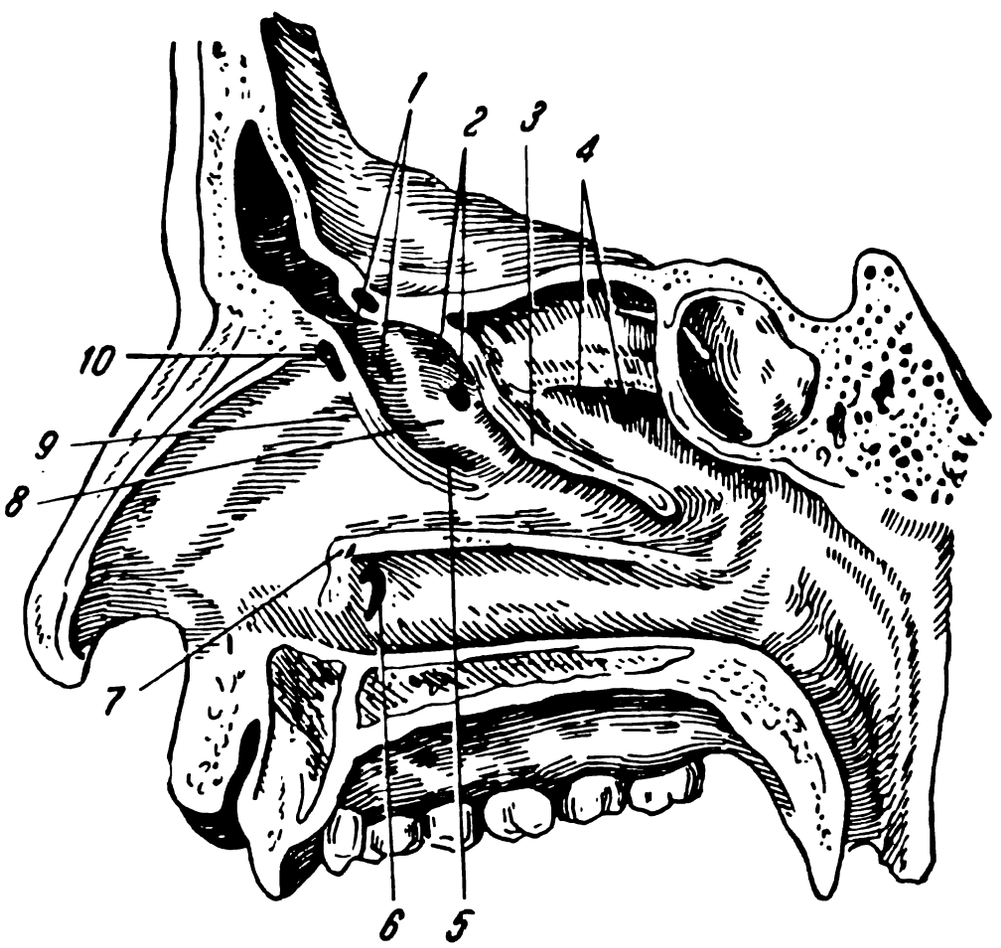


Fig. 38. Lateral Wall of the Nasal Cavity

(1) openings of anterior ethmoid cells; (2) openings of medial ethmoid cells; (3) line of section of middle turbinate; (4) openings of posterior ethmoid cells; (5) opening of maxillary sinus; (6) mouth of nasolacrimal duct; (7) line of section of inferior turbinate; (8) bulla ethmoidalis; (9) uncifrom process (section view); (10) anterior ethmoid cell

the superior, middle and inferior meatuses. The nasal end of the nasolacrimal duct opens below the inferior meatus. The middle nasal meatus has openings to communicate with the paranasal sinuses: (1) the larger, maxillary sinus, or the *antrum of Highmore*, which lies in the body of the maxilla; (2) the *frontal* sinus lying in the squamous portion of the frontal bone, and (3) the anterior cells of the *ethmoid labyrinth.* The posterior ethmoid cells and the *sphenoid* sinus communicate with the superior nasal meatus (Fig. 39).

The cribriform plate of the ethmoid bone forms the roof of the nasal cavity, whose anterior slope consists of the nasal bones and the posterior—of the anteroinferior wall of the sphenoid sinus.

The anterior and posterior parts of the nasal cavity floor consist of the palatine processes of the maxilla and the horizontal plates of the palatine bones respectively.

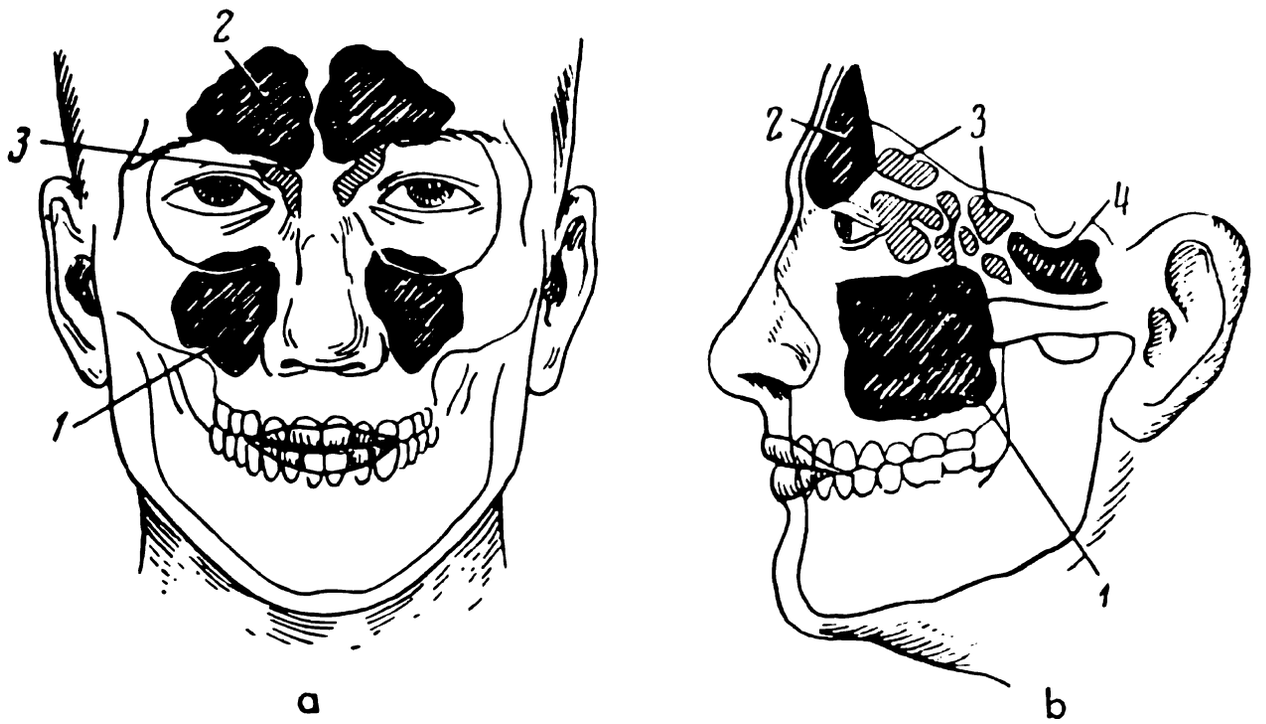


Fig. 39. Scheme of Intercommunication between Paranasal Sinuses (a) front view; (b) lateral view; (1) maxillary (Highmor 's) sinus; (2) frontal sinus; (3) ethmoid labyrinth; (4) sphenoid sinus

The entire nasal cavity is lined with a mucous membrane covered by a stratified, columnar, ciliated epithelium, whose hairs are inclined backwards, towards the choanae.

The mucous membrane of the superior nasal meatus with the adjacent areas of the mucosa of the nasal septum and the upper portion of the middle turbinate is lined with a specific sensory epithelium containing a ramified network of peripheral olfactory nerve endings. This area of the mucous membrane is called the *olfactory membrane*, as distinct from the rest of the nasal mucosa which is lined with a stratified, columnar, ciliated epithelium and is known as the respiratory mucous membrane. The mucous membrane varies in thickness over its area. It is thinnest and most de-

ficient in mucous glands in the paranasal sinuses, and is thickest on the turbinates. Owing to the abundance of a thick meshwork of veins, cavernous or erectile tissue forms in some places of the submucosa which is particularly developed in the inferior nasal turbinate, along the margin of the middle turbinate and on the posterior ends of the middle and superior conchae (Fig. 40).

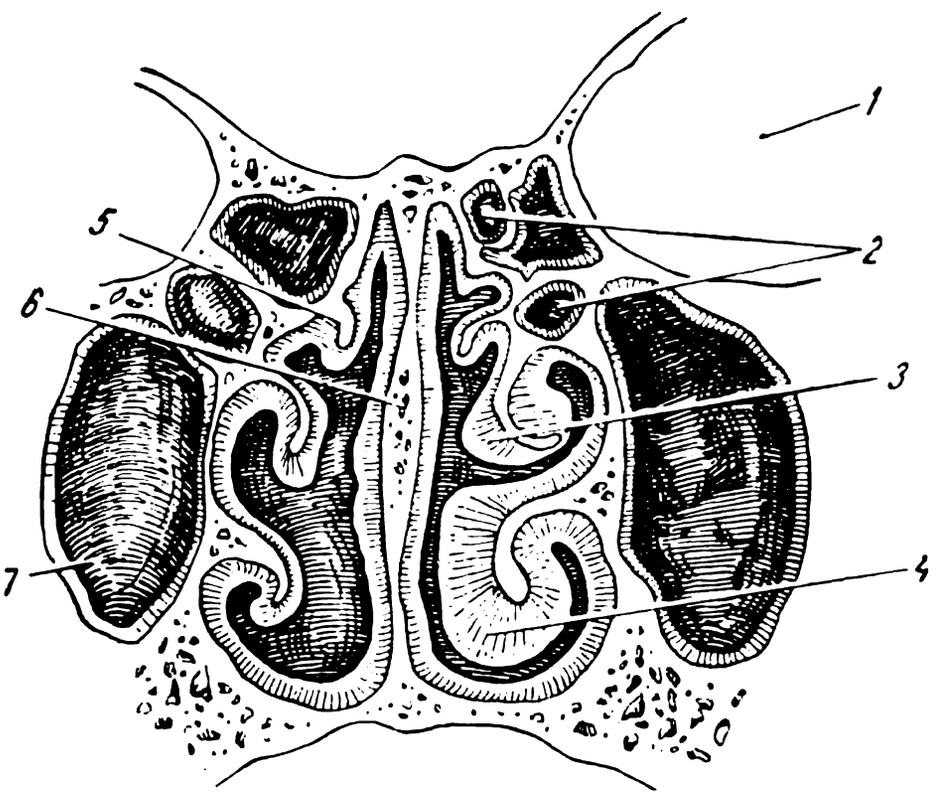


Fig. 40. Nasal Cavity in Vertical Cross-Section
Seen at right is erectile tissue which is considerably swollen

(1) cavity of orbit; (2) ethmoid cells; (3) middle turbinate; (4) inferior turbinate; (5) superior turbinate; (6) nasal septum; (7) antrum of Highmore

The walls of the vessels in the erectile tissue of the conchae are rich in smooth muscle and elastic fibres which enable the erectile tissue to swell and shrink quickly under the effect of various chemical, thermic and mental factors. This is the cause of the sudden fullness in the nose which sometimes occurs.

Vessels of the nasal cavity. The basic arterial supply comes through the sphenopalatine artery which is a branch of the internal maxillary artery. The upper part of the lateral nasal wall receives its blood supply through the anterior

and posterior ethmoidal arteries, which are branches of the ophthalmic artery. Venous blood is collected by numerous minor blood vessels and carried into two major trunks. Part of the venous vessels of the upper nasal region and the superior paranasal sinuses drain directly into the longitudinal sinus of the dura mater.

The mucous membrane of the nasal cavity also has a dense network of lymph vessels which communicate with the subdural and subarachnoid spaces. This fact is one of the important factors contributing to the extension of infection from the nasal into the cranial cavity.

The sensory nerves of the nasal cavity come from the first and second branches of the trigeminal nerve.

The branches of the olfactory analysor enter the nasal cavity through openings in the cribriform plate of the ethmoid bone, and are distributed to specific, highly-differentiated epithelial cells serving as the receptors of odour.

PHYSIOLOGY OF THE NOSE

The nose performs respiratory, olfactory, resonating and defensive functions. Free passage of inhaled air through the nose is an indispensable condition of normal breathing. The air passing through the nasal cavities is largely filtered of its content of inhaled dust particles, warmed to blood heat and moistened when dry. Dust particles and bacteria, as well as larger particles of foreign matter, are caught in the vibrissae of the nasal vestibule as if in a filter. These hairs grow particularly thick in males. The narrowness and irregularity of the nasal meatus ensure close contact between the inhaled air stream and the moist surface of the mucosa, whereby the air is humidified and warmed, and hard particles settle down on the nasal walls. By the action of the epithelial cilia pointing towards the nasopharynx, the particles are carried into the latter with the mucous secretion in which they are enmeshed and then expelled by expectoration or swallowed.

In this way, the air reaching the lungs is filtered of dust to a considerable extent. Experiments performed on people and animals have proved that over a half of the inhaled foreign matter remains in the nose. In mouth breathing,

however, all of the inspired dust gets directly into the pharynx, larynx and deeper into the respiratory tract, where it may cause various morbid conditions.

Nasal breathing has great hygienic advantages over mouth breathing, since the inhaled bacteria which have not been removed with dust are largely rendered harmless and killed by the nasal mucus. The nasal cavity also produces a marked neutralising effect on smoke and toxic chemicals. Complete or partial obstruction of nasal breathing may result in various affections of the lower respiratory tract, such as pharyngitis, tracheitis, bronchitis, etc. The results of nasal obstruction are particularly unpleasant in some fields of industry.

The nasal and nasopharyngeal cavities act as resonating chambers for the voice, wherein sound is amplified by air vibration and the voice acquires timbre and individual sonority. In nasal obstruction, the voice lacks resonance, is muffled and has a nasal twang. This condition is known as *rhinolalia clausa*. If as a result of sagging of the soft palate, due to paralysis, the nasopharynx remains open during phonation, the voice will have a different nasal quality known as *rhinolalia aperta*.

Full or partial obstruction of nasal breathing impairs the sense of smell, and may prevent workers of some specialities, particularly workers employed in the chemical and food industries from fulfilling their duties.

The olfactory sense not only serves to indicate the quality of inhaled matter, but together with the sense of taste, conveys to us the quality of food and drink entering the digestive tract. Moreover, numerous experiments carried out by I. P. Pavlov prove that the sense of smell furnishes a stimulus for reflex secretion of the gastric juices. This stimulus is particularly evident in the reflex secretion of the salivary glands.

Obstruction of nasal breathing interferes with pulmonary ventilation owing to shallow breathing and consequential oxygen deficiency. At the same time it has been found that a longer negative pressure produced by nasal breathing in the deeper portions of the respiratory tract ensures a better pulmonary ventilation with a greater amount of oxygen being absorbed than in the case of mouth breathing.

Mouth breathing results in physical maldevelopment, such as malformations of the chest and the facial bone structure, malocclusion of the teeth, etc.

Procedures Employed in Examining the Nose

Examination of the nose consists in a preliminary inspection of the external nose and examination of the deep-lying parts of the nasal cavity.

In inspecting the external nose, attention is paid to the nasal vestibule. The tip of the nose is lifted upwards, and the patient's head is turned left and right alternately. Examination of the nasal cavity through the nostrils known as anterior rhinoscopy is made by means of a nasal speculum (Fig. 41), artificial lighting and a concave head mirror. The source of illumination should be placed to the right of the patient. The blades of the closed speculum held by the left hand are inserted into the nostril, and then are gently opened far enough to dilate the nostril and bring the nasal cavity into view. The beam of light from the head mirror is focussed within the nasal cavity. The examination should be made according to established routine.

At first, the examination is confined to the lower region of the nasal septum, the inferior nasal turbinate and meatus, and then the attention is turned to the upper region of the nasal septum, the middle nasal turbinates and meatuses, for which purpose the patient's head is tilted back slightly in the appropriate direction. This method of examination is called *anterior*

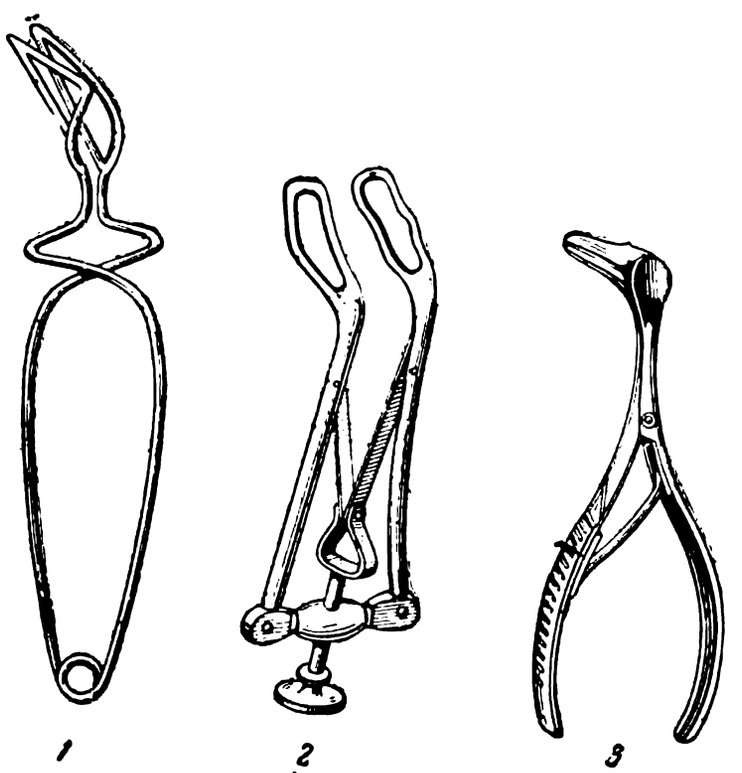


Fig. 41. Types of Nasal Speculums
(1) Stein's speculum; (2) Fränkel's speculum; (3) Hartmann's speculum

rhinoscopy (Fig. 42). For convenience in examining small children, it is advisable to use the aural speculum instead of the nasal type.

Frequently the view of the nasal interior is obstructed by swollen nasal turbinates, in which case vasoconstrictive drugs, such as adrenalin, ephedrine and cocaine, are helpful. A turbinate painted with one of these remedies shrinks considerably, and a much larger part of the nasal cavity thus becomes visible.

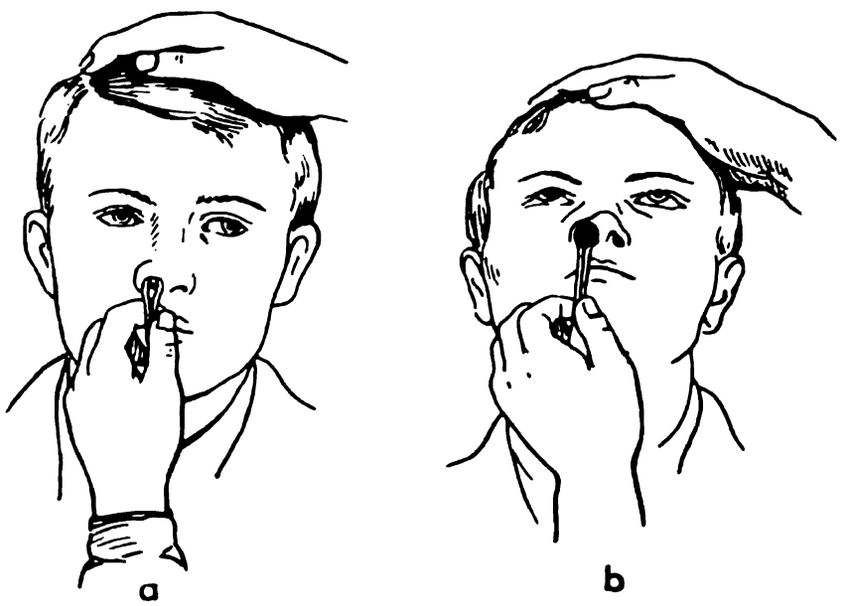


Fig. 42. Anterior Rhinoscopy

(a) examination of inferior nasal meatus; (b) examination of middle and superior nasal meatuses

Should anterior rhinoscopy not facilitate inspection of the posterior region of the nasal cavity, the latter should be examined through the nasopharynx. This method is known as *posterior rhinoscopy* (Fig. 43). The tongue is gently depressed with a tongue depressor, or spatula, and a warmed small postnasal mirror is slipped in over the tongue, until it is behind the soft palate. A spot of light from the lamp is then focussed on the mirror, and the latter will show a reflected image of the posterior nasal cavity and part of the nasopharynx. The mirror should be inserted into the nasopharynx without touching the soft palate, the palatine arches, the tongue and the posterior wall of the pharynx, as it may cause the patient to gag. The mirror will reflect the posterior edge of the vomer, with the choanae on both sides and the posterior ends of the inferior, middle and, sometimes, superior conchae lying in their gaps. In addi-

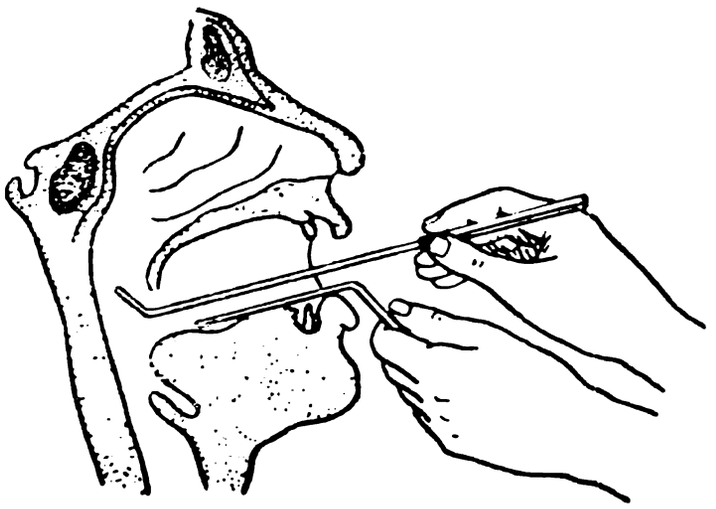


Fig. 43. Posterior Rhinoscopy

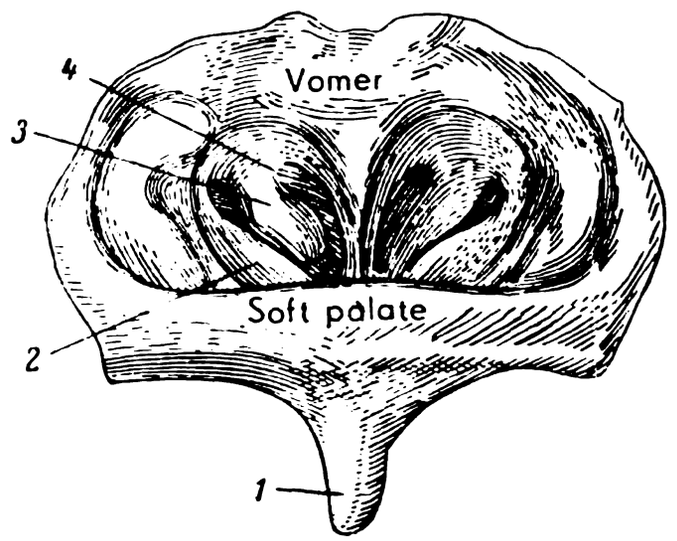


Fig. 44. View of Choanae in Posterior Rhinoscopy

(1) uvula; (2) inferior turbinate; (3) middle turbinate; (4) superior turbinate

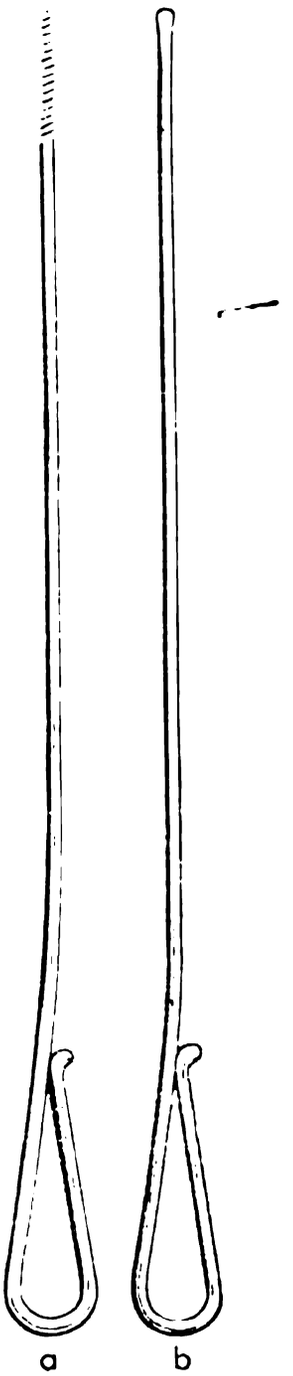


Fig. 45. Nasal Probes

(a) threaded probe; (b) eyed probe

tion, the posterior surface of the soft palate, the vault of the nasopharynx, and the pharyngeal openings of the auditory tubes may also be examined (Fig. 44).

The chief difficulty likely to arise in posterior rhinoscopy is that the patient may have a hypersensitive pharynx and choke easily. This is especially the case with small children.

In such cases the pharynx is painted with 2-3% cocaine solution, while in the case of children the finger may be used to palpate the nasopharynx. This examination is made with the right forefinger and with another finger pressing the flesh of the child's cheek between its teeth to keep it from biting (see Fig. 69, p. 165).

The results of examination of the nose should be verified by probing. The latter is also used to determine the consistency of the mucosa and the inflamed tissue or tumour. The probing of the nasal cavity in adults is made with an eyed nasal probe (Fig. 45, a and b); in a hypersensitive patient, this should be preceded by painting his mucous membrane with 2-3% cocaine or 1% dicaine solutions.

Assessment of the nasal function consists in checking the passage of air through the nasal cavity and the acuity of the sense of smell. The patency of the nasal cavity can be easily checked by directing the patient to take a deep breath and exhale the air through one nostril at a time. If the passage is free, a piece of loose cotton wool held under the nostril will flutter.

For practical purposes the acuity of smell is determined by using various odours, as that of 0.5% acetic acid solution with a weak smell, spirit of wine of moderate odour, a simple valerian tincture with a strong smell, camphor and oil of cloves which have an extremely strong smell.

When testing the sense of smell, one nostril is jammed with a finger, and the other is left open to smell a piece of cotton wool or filter paper soaked in one or another solution. First come solutions of weaker smell, and then stronger solutions are used, if the patient fails to perceive the former.

GENERAL METHODS OF TREATMENT IN NASAL DISEASES

The *painting* of the nasal mucosa with cocaine or dicaine for anesthesia or with a medicinal solution for therapeutic purposes is made with a cotton applicator under the guidance of vision.

The *insufflation* into the nose of various powder drugs as part of conservative treatment or following an operation is carried out with various types of insufflators.

The *cauterisation* of the nasal mucosa with silver nitrate, trichloracetic and chromic acids is performed for therapeutic purposes, as well as for the arrest of hemorrhage. Prior to cauterisation, the nasal mucosa should be painted once or twice with 3-5% cocaine solution.

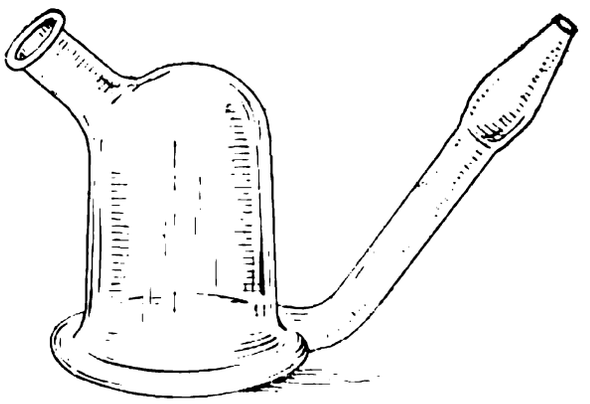


Fig. 46. Nasal Washing-Bowl

Nasal douching should be carried out with great care, and the patient should be told in detail how this is done. A. F. Ivanov, a Soviet practitioner, has suggested a method in which the solution is administered with a tea spoon, the patient lying down with his head propped high on a pillow. The solution should be poured in slowly to avoid choking, as otherwise the adhering liquid may be pushed into the Eustachian tube. The patient should be warned of this in advance.

Nasal douching can also be carried out with a special glass washing-bowl (Fig. 46) containing water or a medicinal solution. Only sterile water should be used; and medicinal solution should first be dissolved in distilled or boiled water. Taking the bowl by the right hand, the patient closing its rear tube with a forefinger insert the front tube fitted with a nasal tip into one nostril. By lifting the forefinger the fluid will be allowed to flow and wash the nasal cavity and then be collected in a basin held below the nose. A small quantity of fluid is syringed into the nose by alternately opening and closing the rear tube. The patient should be warned to hold the head somewhat bent forward during the process. The liquid should be warmed to body heat to prevent the subsequent onset of headache. The patient should be warned against swallowing during the douching, as otherwise the liquid could be forced into the Eustachian tube. When blowing the nose after a douche, the patient should have one nostril and the mouth open to keep down pressure in the nose and nasopharynx, and ward off the danger of the nasal secretion being forced into the middle ear through the auditory tube.

A safer method of nasal douching is by means of a special sprayer described in the chapter on general technique in the treatment of the larynx. The sprayer tip is inserted into the nose in such a manner that the liquid pulverised by periodical compression of the sprayer bulb primarily floods the inferior nasal meatus.

The nasal cavity may be cleansed by the same method used in douching but if crusts are present in the nasal vestibule, a piece of cotton wool greased with liquid or soft petrolatum is inserted into the nose for 15 to 20 minutes to soften the small crusts and remove them with a pair of pincers. If there are crusts closely adhering to the mucosa in the deeper parts of the nasal cavity, a special tamponade will be needed. A piece of cotton wool is inserted into the nasal cavity on a probe and allowed to remain for a quarter to half an hour or more. This will stimulate abundant secretion of the mucous membrane causing the crusts to break off readily. The latter can then be removed by blowing the nose or with instruments. Drops warmed to blood heat are instilled into the nose with a medicine dropper in portions of 1, 2, 5 drops, the head being tilted slightly back and the nose tip lifted with a finger.

The nasal cavity is lubricated with oil by means of cotton wool held on a threaded probe, from which it is later removed with a pincers or a piece of dry cotton wool.

Surgical interference on the nasal cavity is performed by galvanocautery, diathermocoagulation, and special surgical instruments.

In *galvanocautery*, the following rules should be observed: (1) the cauter should not be heated to white heat, since the hemostatic effect is produced only by a red-hot cauter; (2) care should be taken to avoid simultaneous burning of both walls of the nasal cavity and their subsequent accretion.

In operations on the nasal cavity, it is impossible to *sterilise* the operative field, as can be done on the skin. The surgeon must content himself with cleansing the nasal cavity of pus and mucus by lightly wiping it with sterile cotton applicators.

Experience shows that thanks to an efficient blood supply wounds in the nasal cavity heal readily even without any

careful disinfection of the mucous membrane, provided general surgical rules are strictly adhered to, including thorough sterilisation of surgical instruments and other material, as well as the operator's hands.

Surgical operations on the nasal cavity are performed with various instruments shaped to fit the nasal cavity and its various structures.

One of the chief difficulties in intranasal operations is nasal bleeding. Therefore, prior to the operation it is important to check whether or not the patient has poor blood clotting by testing his blood for coagulation. Moreover, operations on patients with anemia, chlorosis, leukemia, renal diseases, etc., should be made with great care. Local cocaine and adrenalin anesthesia helps considerably to reduce hemorrhage.

In most cases, operations on the nasal cavity are performed under *local anesthesia* in the soft, as well as in the hard, bony portions. To this end, the mucous membrane is painted with 5-10% cocaine solution or a 1%, 2% or 3% solution of dicaine with adrenalin or ephedrine. A drop of adrenalin solution, 1:1,000, is added to every milligram of cocaine solution to drain the mucosa of blood still more, and simultaneously slow down cocaine absorption. In operations on soft tissues two or three paintings are usually enough, whereas in more extensive operations, for instance on the ethmoid labyrinth or in resections of the nasal septum, application of a small gauze strip soaked in cocaine solution is recommended for a short period.

In operations on the nasal septum, 0.5% novocaine-adrenalin solution may be injected into the mucous membrane.

General anesthesia is rarely used in operations on the nasal cavity, in fact, only in some cases of removal of malignant tumours and in extraction of foreign bodies in small children.

Postoperative treatment of the nose or nasal cavity includes checking the pulse rate and general condition of the patient and changing blood-soaked dressings.

The dressing is a wide gauze strip with its ends cut or torn to tie up cotton wool inside and fixed round the head.

Burns. Burns of the nasal skin are commonly part of a facial burn, mostly of the first degree, caused by exposure to sunlight, contact with flame or excessive irradiation with a quartz lamp. The latter produces uniform redness over the skin attended with pain which intensifies in a few hours' time and subsides within three to five days. By this time, the tension in the skin lessens as well as the swelling present in more severe burns. Erythema quickly disappears, and the skin takes on a light brown tinge known as tan. Four to five days later, the horny layer of the epidermis begins to scale off.

Treatment. The strained feeling and itching in the skin can be somewhat relieved by rubbing it lightly with white petrolatum, lanolin or zinc ointment.

A beneficial effect is produced by ointment with the following formula:

Rp. Dermatoli 1.5
Lanolini
Vaselini aa 7.5
M. f. ung.
DS. Ointment

Wet dressings of Burow's solution or neutral powders, starch, talc, and zinc oxide are also helpful. Prophylaxis in case of prolonged exposure to sun-rays is by painting the open skin with petrolatum or some other mild ointment.

Frostbites. The nose is less susceptible to freezing than the auricle. Frostbites of the nose mostly occur in weak, anemic subjects. The nasal skin becomes red and sometimes bluish at the nasal tip. There is itching in the affected areas and a rather painful burning sensation upon entering a warm room. More severe freezing may later lead to infiltration of tissues, necrosis and deep ulceration, although such cases are exceedingly rare.

Treatment. The chief effort should be to remove the basic cause of the condition and prevent its recurrence by appropriate body hardening. Light frostbites are treated by a quarter-hour application to the nose of hot, wet dressings of a weak Burow's solution and bland ointment, as well as by using bandages lubricated with lard or goose fat.

Very severe freezing resulting in necrosis and ulceration will require surgical interference.

Nasal erysipelas (*erysipeles nasi*). This disease is presumably of streptococcic etiology, and affects the face and skull in seven out of every ten cases. The erysipelatous inflammation of the nasal mucosa usually occurs by extension of facial erysipelas. Primary nasal erysipelas may be observed in case of skin abrasions and chaps at the nasal vestibule. The disease usually starts with a rigor, sudden fever, severe malaise and acute headache. Other classical symptoms are marked tenderness and bright redness which spreads irregularly over the face within sharply outlined areas. Skin fissures frequently occur around the nasal wings. Rhinoscopical examination will reveal a markedly red and tender nasal mucosa, sometimes covered with blebs. Regional lymph glands become swollen on the very first day of the disease. A change in reactivity of the body and impairment of its immunological properties may account for the so-called habitual or relapsing erysipelas in empyema of the paranasal sinuses, as well as for erysipelas of the pharynx and nose in chronic inflammations of the pharyngeal tonsils. Erysipelas in mucous membranes is often accompanied by a most severe malaise and may cause thrombophlebitis of the veins or meningeal inflammation.

Diagnosis. The diagnosis is usually easy to make, as nasal erysipelas is accompanied or preceded by a similar condition on the face.

Treatment. An excellent remedy is penicillin which should be administered in 100,000 unit doses six times in 24 hours. Even more effective are synthomycin and levomycetin which quickly eliminate toxicosis and bring down body temperature on the first day of treatment. The drug is taken orally in a 0.5 g dose four times in 24 hours for a period of five to seven days. Another good remedy for erysipelas is streptocide whose single dosage for an adult patient should be 0.5 g to be repeated five to six times in 24 hours.

In addition, it is recommended that the inflammatory focus be exposed once or twice to ultraviolet light in an erythema dose. Local treatment is by application of an aseptic dressing of camphor oil to relieve the painful tension.

Prophylaxis. Prevention of nasal erysipelas is by the treatment of empyema of the paranasal sinuses, as well as abrasions and excoriations around the nasal vestibule, as the latter may be scratched raw and lead to infection causing relapsing erysipelas. It is desirable to isolate erysipelatous patients.

Diseases of the Nasal Vestibule

Eczema of the nasal vestibule (*eczema vestibuli nasi*). Eczema frequently accompanies chronic coryza, suppuration in the paranasal sinuses, and diseases of the pharynx. Frequent blowing, wiping and picking of the nose will make this condition persist for a long time.

In acute cases, there is redness, swelling and scaling of the skin around the nasal vestibule; sometimes, there are eruptions, crusts and most painful fissures in the corners of the nasal vestibule, which are ringed with inflamed tissue. All these symptoms are accompanied by intense itching.

In children, particularly those with exudative diathesis, the upper lip and the corners of the mouth are prone to eczema in the constant presence of an irritating nasal discharge. The upper lip is swollen and covered with lamellar scales and crusts, and sometimes with tiny vesicles which burst readily, leaving eroded surfaces.

In chronic cases, inflammatory symptoms are less pronounced, and the entire nasal vestibule is covered with fairly dense scab formations. A prolonged eczema of the nasal vestibule may provoke pyogenic infection with an open danger of a furuncle, erysipelas and sycosis, as well as a general septic disease, though the latter is less frequent.

Treatment. Its purpose is to eliminate the basic cause of the condition, i.e., diseases of the nose, nasopharynx, and paranasal sinuses accompanied by a purulent discharge. For children, adenoidectomy is commonly employed to root out the cause of the disease and aid recovery.

When necessary, the patient's general condition may be improved by administration of arsenic and ferrum drugs, vitamins and cod-liver oil, as well as by suitable climatic and sanatorial treatment. Local applications are used to remove crusts and cleanse the skin; the best method is to pack

the nose twice a day for half an hour with a cotton tampon soaked in vegetable oil or a warm baking soda solution. The softened crusts can be removed with ease. Forcible removal of dry crusts is not only painful, but will cause bleeding and new crust formation. The crusts may also be picked off after application of 2% salicylic ointment. Following crust removal, the raw areas should be lubricated with boric, zinc or white mercury ointments. The following ointments are effective:

Rp. Mentholi 0.1
Protargoli 0.2
Vaselini 10.0
M. f. ung.
DS. Nasal ointment

Rp. Bismuthi subnitrici 3.0
Vaselini albi
Lanolini aa 15.0
M. f. ung.
DS. Nasal ointment

Rp. Zinci oxydati 5.0
Lanolini
Vaselini albi aa 10.0
M. f. ung.
DS. Nasal ointment

Rp. Ung. Hydrargyri praecipitati albi 2% 15.0
DS. For external use. Nasal ointment

Rp. Pastae Lassari 15.0
DS. Lubricate the nasal vestibule twice daily

Acute weeping eczema can be dried up effectively with frequently changed wet dressings of 1 to 2% resorcinol solution or 3 to 5% resorcinol ointment.

Sycosis of the nasal vestibule (*sycosis vestibuli nasi*). This is a staphylococcus infection of the hair follicles in the nasal vestibule.

The initial symptom is the eruption of bright-red, solid nodules, the size of a pin-head, with hair-pierced pustulae which quickly develop at their apex. The latter's purulent contents quickly dry into a yellowy or light-brown crust tightly adherent to the hair stem.

Sycosis is caused by staphylococci present in a purulent nasal discharge or introduced on soiled fingers. This disease very often accompanies eczema, and where there are unbroken crusts and infiltration, it is difficult to differentiate it from the latter.

Treatment. The hairs are removed from the pustulae with pincers. As the disease is stubborn, this procedure must be repeated. At first, the crusts are usually softened and removed, the skin is then sterilised by wiping it with 1% salicylic alcohol on cotton wool, and, finally, it is lubricated with bland ointments. Dense infiltrations often present in sycosis may be resolved by the use of hot, wet dressings of potassium permanganate solution, as well as with 1% to 5% synthomycin emulsion, ultraviolet light and Bucky's rays. Penicillin is also recommended in an overall dose of 1 to 1.5 million units to be injected in 100,000 unit portions every three hours during the day, as well as staphylophage and autohemotherapy. The normal skin is wiped with 1% camphor oil on cotton wool.

The disease is very apt to recur.

Furuncle of the nasal vestibule (*furunculus vestibuli nasi*). This condition which is often associated with general furunculosis is brought about by scratching and the resulting entry of staphylococci into the outlet ducts of the sebaceous glands and the hair follicles in the nasal vestibule.

The tip or wing of the nose becomes markedly red, tense and tender. The inflammatory redness and swelling may spread to the surrounding parts of the face. Close examination of the most inflamed area of the nose will reveal a cone-shaped infiltration and redness. This place is the most tender to touch.

Sometimes an abscess forms at the site of infiltration, and the temperature may considerably rise.

Treatment. The treatment is conservative by the application of bland ointments or by packing the nose every three to four hours with Burow's solution on gauze pledgets. At the time of extension of the disease with a more or less high fever and growing inflammatory swelling, penicillin or streptomycin should be administered in full dose. Under no circumstances may any form of squeezing and evacuation of the boil be attempted, since this can lead to serious complications. These complications may develop by extension of the infection from the nose through the veins into the cranial cavity. In lingering furunculosis, general irradiation with a quartz lamp, proteinotherapy and autohemotherapy will aid.

Diseases of the Nasal Cavity

Synechiae and atresias of the nasal cavity. A major part of abnormal conditions requiring medical attention is made up by congenital and acquired adhesions of parts in the nasal cavity as well as deflections of the nasal septum. The adhesions fall into two types: (a) *synechiae* which for the most part are thin scar-tissue fibres connecting separate parts of the nasal cavity walls, and (b) *atresias*, or conditions where the nasal passage is blocked either completely or partially. Adhesions may be made of cartilage, bone or scar tissue.

In the majority of cases, these adhesions follow nasal ulceration caused by acute infectious diseases, such as smallpox, diphtheria, typhoids, etc. Deformities in the nasal cavity and nares may also result from lupus, syphilis and rhinoscleroma, less frequently from trauma, as well as from chemical or galvanic cautery of the nose made accidentally or therapeutically. Various kinds of adhesions can be easily detected by anterior and posterior rhinoscopy as well as by probing the nasal cavity.

Treatment. The treatment is surgical by cutting the adhesive fibres with a scalpel or galvanocauter and by providing for rubber or celluloid partitions between the separated tissues until healing takes place.

Sometimes, a more complicated surgical operation may be required.

Nasal Hemorrhage (Epistaxis)

The causes of frequent epistaxes may be of local or systemic origin. The latter refer to diseases of the blood, atherosclerosis, diseases of the heart, kidneys, liver, etc. The febrile condition and vascular changes peculiar to many infectious diseases are very likely to produce copious hemorrhage, as for example in influenza, enteric and relapsing fever, scarlet fever, smallpox, and also in malaria. Finally, blood congestion in the head, tumours on the neck, and whooping cough may also lead to nasal hemorrhage. Bleeding from the nose is fairly common in hypertension or may be its first indication. The local causes of nasal hemorrhage are

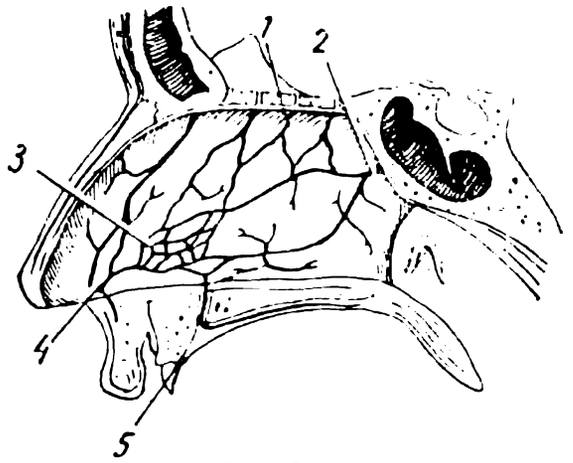


Fig. 47. Vessels of Nasal Septum
(1) *a. ethmoidalis;* (2) *a. nasalis posterior;* (3) vascular region of nasal septum, most frequent site of epistaxis; (4) anastomosis with *a. labialis* superior; (5) anastomosis with palatine arteries through *canalis incisivus*

most frequently nasal trauma, picking the nose, injuries to the nasal mucosa during removal of hard crusts, bleeding tumours and ulcerations in the nose. Sometimes, hemorrhage follows an operation in the nose and nasopharynx.

The most common site of nosebleed (Fig.47) is the anterior part of the nasal septum, approximately 1 cm above the nasal orifices. Bleeding caused by atherosclerosis mostly occurs from a point in the posterior region of the nose. The mucosa of the nasal conchae or other parts of the nose is less likely to bleed. Bleeding is usually of sudden onset without any obvious cause. Fairly often, it ceases spontaneously, but sometimes it goes on for a long time draining the patient of blood, slowing down the pulse rate, causing constitutional debility and fainting.

Treatment. First aid in nasal bleeding is to put the patient in quiet surroundings, undo his collar and belt and forbid him to make any abrupt movements and blow the nose. In simple cases, it is enough to put the patient in bed with his head propped high on two or three pillows and instruct him to press the nasal wing to the septum to stop the bleeding. If this method fails, a pledget of sterile cotton wool and gauze, either dry or soaked in a hydrogen peroxide or warm thrombin solution, is placed anteriorly against the bleeding area. For some time, the patient keeps this pledget compressed against the nasal septum. Light hemorrhage can easily be checked in this way. A more effective method, particularly in recurrent hemorrhage, is to locate the bleeding points with a head mirror and nasal speculum, paint them with a 5% solution of cocaine and adrenalin and cauterise them with chromic acid, trichloracetic acid or with a galvanocauter.

The anterior tamponade of the entire nasal cavity is made with the aid of an angular or crocodile forceps (Fig. 48)

used to widen the nasal vestibule and a head mirror to illuminate the nasal cavity. A gauze ribbon tampon is consecutively and firmly packed into the posterior departments of the nasal cavity, the inferior and middle meatuses and the nasal vestibule (Fig. 49, *1*).

Should all these measures fail, the patient must immediately be hospitalised. In exceptional cases, particularly after an operation in the nose and nasopharynx, as well as in nasal traumas, all these methods are inadequate to control the hemorrhage, and the nasal cavity is packed by means of postnasal, or posterior, tamponade (Fig. 49, *2*, *3*, *4*).

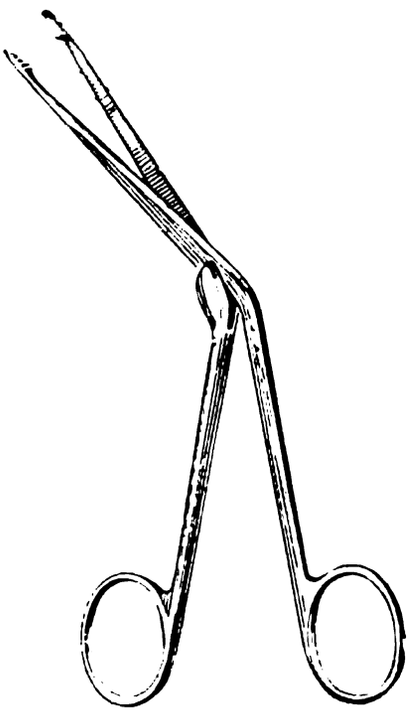


Fig. 48. Dressing Forceps

A thin rubber catheter is passed through the inferior nasal meatus into the nasopharynx. When its end appears behind the soft palate, it is seized with forceps and drawn outside through the mouth. A gauze tampon cut to the size of the nasopharynx is prepared in advance and tied up crosswise like a bale with a strong double silken thread, whose three long ends are left free. After the tampon has been tied with two threads to the seized end of the catheter, the latter is brought back through the nasal passage with both threads following through one of the nostrils. The plug is then inserted by the finger into the nasopharynx and drawn up into its roof, where it is anchored by the threads passing through the nostrils which are kept taut, while the nasal vestibule is packed in the usual way, and they are then tied over a cotton pad below the nostril. To avoid ear disease the tampon is removed from the nasopharynx one or two days later. It is pulled out by the third thread.

In severe hemorrhage, chemotherapy is recommended to promote blood coagulation, with 10% calcium chloride solution to be taken orally.

Rp. Sol. Calcii chlorati 10% 200.0
DS. One tablespoonful dose three to four times daily after meals

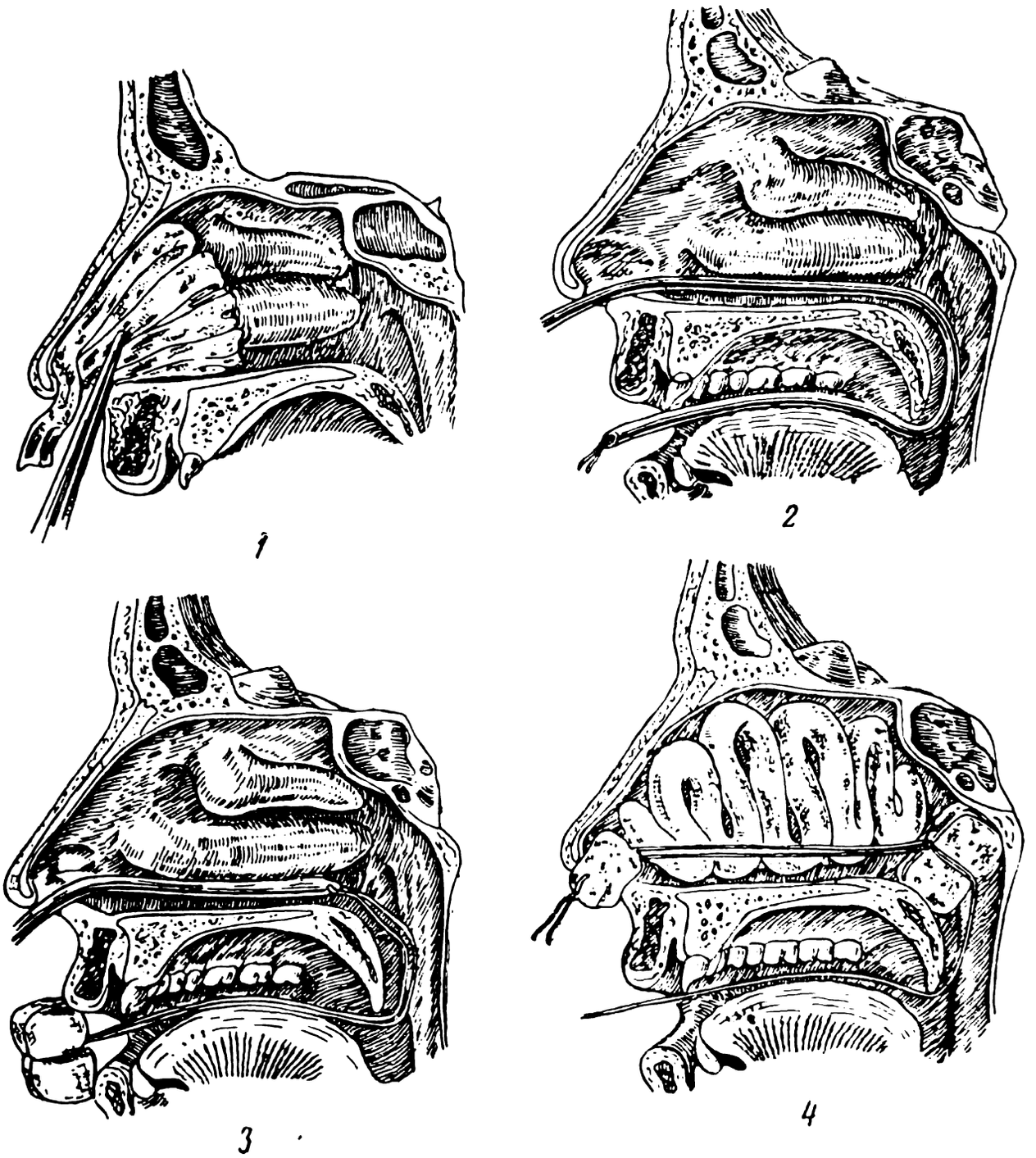


Fig. 49.

(1) anterior nasal tamponade; (2) first stage of posterior tamponade; (3) second stage of posterior tamponade; (4) concluding stage

Another drug is a vitamin K preparation, vikasolum.

Rp. Vikasoli 0.01
D. t. d. N. 10
S. One pill twice a day

Vikasolum may be given by intramuscular injection.

Rp. Sol. Vikasoli 0.3% 5.0
D. t. d. N. 6
S. Dose of 5 ml intramuscularly

Should these drugs be ineffective, blood transfusion in fractional doses of 50 to 100 ml may be used. A cold, wet dressing may be applied to the nasal bridge and an ice bag to the nape.

Foreign Bodies in the Nasal Cavity and Rhinoliths

Foreign bodies in the nasal cavity are most common in children who insert into the nose at play various small objects such as beads, peas, cherry stones, sunflower seeds, buttons, etc. In adults, foreign bodies in the nasal cavity may be the result of trauma or a gunshot wound. Calcareous masses, known as rhinoliths sometimes form in the nasal cavity owing to salts depositing themselves around a foreign body. Rhinoliths may be of various shapes and sizes, sometimes forming a sort of a cast of the nasal cavity.

Foreign bodies are usually to be found in the inferior meatus, near the nasal vestibule, whence they may be pushed deeper by an unskilful attempt at removal. Smooth foreign bodies and rhinoliths cause no pain. In some cases, foreign bodies obstruct only one side of the nose; in other cases, however, they produce a copious and usually foul discharge owing to inflammation of the nasal mucosa. A unilateral purulent nasal discharge in children is indicative of a foreign body in the affected side of the nose.

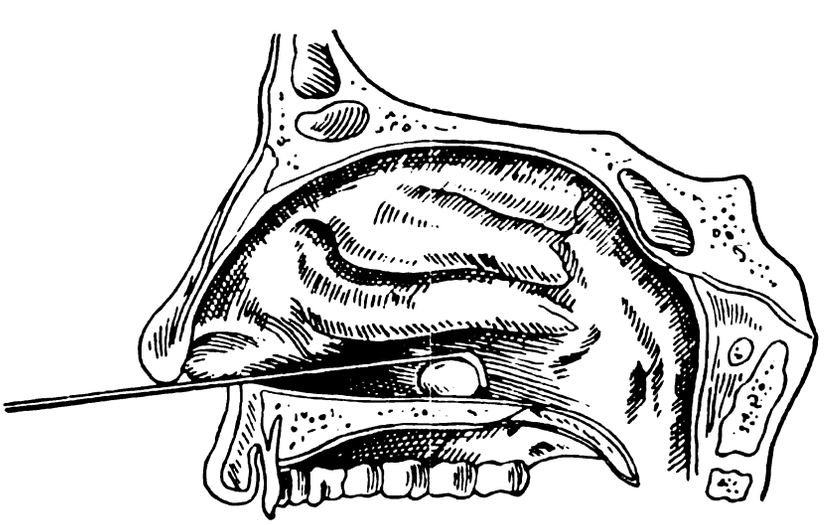


Fig. 50. Hooking Out Foreign Body from Nasal Cavity with Blunt Probe

When removing a foreign body in a child, the latter should be firmly wrapped in a sheet like in adenotomy.

The foreign body is removed after careful anesthesia and decongestion of the nasal mucosa have been carried out by spraying it with cocaine or dicaine solutions mixed with adrenalin or ephedrine. Following decongestion the child should be told to blow the nose through the passage containing the foreign body. If the child is very restless, a short general anesthesia may be required. The foreign body is hooked out with a blunt probe passed and turned behind it under the guidance of vision (Fig. 50).

Diseases of the Nasal Septum

Deviation of the nasal septum (*deviatto septi nasi*). In adults, the nasal septum rarely follows the midline. More commonly, it is displaced from the vertical to one side or to the other. These deflections are of various shapes (Fig. 51) and may be found both in the anterior and the posterior parts of the nasal septum, though they are much rarer in the latter.

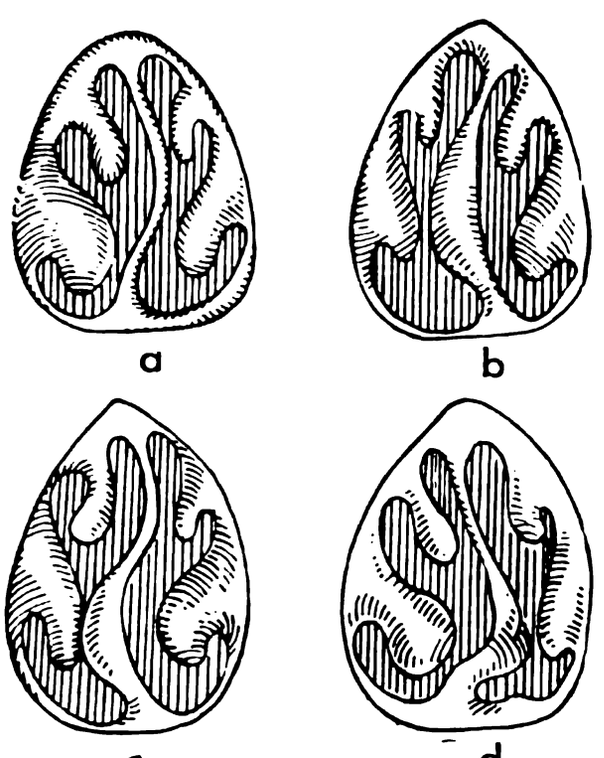


Fig. 51. Deflection of Nasal Septum

(a) slight deviation; (b) deviation with swollen mucosa; (c) S-shaped deviation; (d) angular deviation with swollen mucosa

Apart from simple deviations, the nasal septum may often have bony outgrowths known as spurs and ridges. These projections often combine with deviations from the midline and are located on the convex side of the nasal septum, mostly at the site where the cartilage is attached to the vomer.

Symptoms. The basic symptom of septal deviation is nasal obstruction in one or both sides of the nose.

Diagnosis. This is easily made by anterior rhinoscopy.

Treatment. A septal deformity can only be corrected by

surgery. The indication for surgical interference is obstruction to proper nasal respiration in one or both sides of the nose.

Submucous resection of the septum is performed in the following manner. Following anesthesia an incision is made in the mucous membrane and perichondrium and carried down to the cartilage on the convex side. The mucous membrane with the perichondrium covering the deformed part of the nasal septum is elevated from the underlying cartilage, and the latter is cut through in the line of the first incision, care being taken not to perforate the mucosa on the opposite side. Through the incision in the cartilage, the mucosa with the perichondrium is elevated on the other side of the septum, after which the deformed part is removed with retention of a 1 cm wide cartilaginous plate at the nasal dorsum to prevent formation of a "sunken nose". Following the removal of the deformed bone and cartilage from the nasal septum, both layers of mucosa and perichondrium are again placed together and kept in this position for 24 hours by means of nasal packing for both nostrils. The flaps of nasal mucosa stick together and heal in a few days' time. After removal of the packs, the nasal airway is not restored to its full capacity at once because of the presence of a reactive inflammatory swelling in the mucosa. In place of the deviated septum of bone and cartilage a straight nasal septum largely formed of membrane develops. Owing to the need for nasal packing, this operation is liable to complications, such as tonsillitis and acute otitis media.

The instruments used for submucous resection of the nasal septum are shown in Fig. 52. The Soviet surgeons V. I. Voyachek and M. F. Tsytovich have suggested a new, economical operation on the nasal septum, whereby the latter can be made to fall in the midline without removal of its basal structure of bone and cartilage, in which case the procedure is mobilisation and redressment, or with removal of a thin ring-shaped piece of cartilage known as circular resection.

Hematoma and abscess of the nasal septum (*haematoma et abscessus septi nasi*). Hematoma is a frequent result of external injuries causing hemorrhage under the perichondrium of the nasal septum. If a hematoma is not opened in time it nearly always turns into an abscess as a result of secondary infection. An abscess may occur in cases of nasal

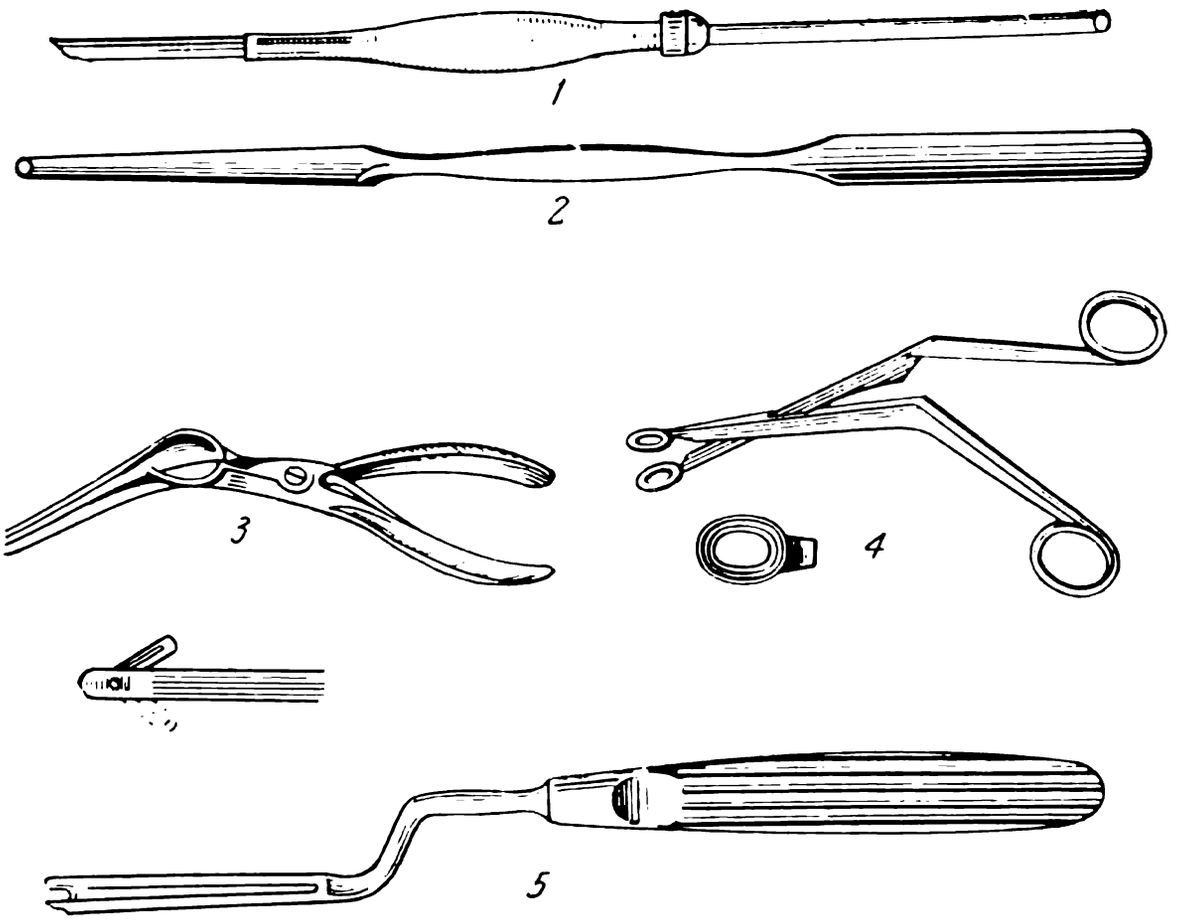


Fig. 52. Instruments for Submucous Resection of Nasal Septum
(1) mucosal dissector; (2) raspatory elevator; (3) Killian's speculum; (4) nasal forceps; (5) trephine

erysipelas, caries of the incisor roots or infectious diseases, by metastasis. Rhinoscopy and sometimes lifting the nasal tip with a finger will show bag-like bright-red swellings on both sides of the septum which pit readily if touched with an eyed probe. Abscess formation in the nasal septum is commonly characterised by a rise in temperature, marked subjective symptoms; headache and severe nasal obstruction. If the diagnosis is in doubt, the abscess should be punctured and its contents drawn into a syringe.

Treatment. The cure of hematoma and abscess of the nasal septum is by free surgical incision, sometimes on both sides of the septum, in which case the incisions should be made at different levels. This operation may be entrusted to an assistant. Aftercare is by draining the abscess with packs introduced through the incision. A belated opening of the abscess may result in saddle-nose deformity owing to a partial destruction of the quadrangular cartilage.

ACUTE INFLAMMATIONS OF THE NOSE

Acute rhinitis (*rhinitis acuta*). Acute rhinitis, otherwise known as acute coryza or the common cold, is one of the most common infections of the upper respiratory tract. It may occur independently or as the prodromal symptom of an acute infectious disease. Infection is the most common cause of acute coryza. Chilling of the body makes it susceptible to germs. Among acute infectious diseases accompanied by the common cold is influenza, where the upper air pas sages (nasal cavity) are often the basic or primary focus of the disease, as well as measles, scarlet fever, diphtheria and erysipelas. The course of the common cold is usually found to comprise three stages. The onset is marked by a hot and dry feeling in the nose, sneezing and fever of upwards of 37°C. The nasal mucosa is red and dry.

A few hours, or sometimes two to three days, later, the clinical picture changes, the mucous membrane becomes swollen and sodden, and a very copious watery secretion begins to drip from the nose. At the same time, the extremely obnoxious feeling of tension in the nose and nasopharynx is relieved. These symptoms refer to the second stage.

The nasal discharge then turns mucoid and as a result of an admixture of shed epithelium and leukocytes, becomes purulent. The nasal discharge thins out, the mucous membrane begins to heal rapidly, and full recuperation follows in one or two weeks' time.

In its first stage the common cold is apt to produce a heavy feeling in the head and headache. If later there is an extension of the inflammation to the frontal and Highmore's sinuses, painful sensations may occur in the region of the forehead and eye-socket. Nasal obstruction may often affect the timbre (sonority) of the voice and give it a nasal quality. Sometimes it causes a decreased sense of smell.

The common cold is very liable to complications in the eye conjunctiva, whose symptoms are reddening of the eyes and excessive lacrimation. Extension of the infection through the Eustachian tube into the middle ear frequently causes catarrhal or purulent inflammation in the latter. Nasal discharge, particularly in children is apt to macerate the

skin of the nasal vestibule which becomes swollen, chapped and tender.

Treatment. At the onset, there is hope of halting the common cold by giving the patient hot tea, sudorific and antifebrile drugs. Phenacetin in a 0.3-0.5 g dose, acetylsalicylic acid in a 0.5-1 g dose, and other drugs are given for headache. Symptomatic treatment is to eliminate nasal obstruction, if only for a short period, which brings temporary relief and removes disorders associated with inadequate nasal respiration. This is achieved by the use of cacaine with adrenalin or menthol given in the nose by drops or in ointment. The following formulae are particularly recommended:

Rp. Cocaini hydrochlorici 0.15
Sol. Adrenalini hydrochlorici (1:1,000) gtt. V.
Sol. Acidi borici 3% 10.0
MDS. Five drops in each nostril four to five times daily

Rp. Mentholi crystallisati 0.15
Acidi borici pulverati
Zinci oxydati $\overline{aa}$ 0.75
Vaselini flavi 15.0
DS. Nasal ointment

Rp. Mentholi crystallisati 0.05
Ol. Amygdalarum dulcium 10.0
MDS. Five drops in each nostril twice daily

Hydrochloric ephedrine has a more durable vasoconstrictive effect than cocaine and adrenalin. It is used as a solution, 2 or 3%, given in drops or ointment.

An effective remedy is sanarin, a drug of Czechoslovak origin, which is given by drop instillation or in emulsion.

The sufferer from the common cold may be helped considerably by nasal insufflation of sulfonamides (white streptocide, sulfadimezin in equal weight portions) in powder form combined with penicillin in a 200,000 unit dose.

The common cold in babies. This disease presents a special danger to babies. The nasal meatuses in infants are very narrow, and nasal obstruction is very likely to follow even a minor swelling of the mucosa.

Apart from disorders due to the absence of nasal respiration, such as excitability, broken sleep, etc., nasal ob-

struction may often lead to emaciation of the baby, which is unable to suck at the breast normally.

Infectious nasopharyngitis disturbs the child's appetite, and its swallowing the toxic nasopharyngeal secretion may lead to dyspepsia.

Sometimes, the inflammation in the nasal mucosa extends to the mucous membranes of the nasopharynx and auditory tube with resulting acute otitis media, or it spreads down the respiratory tract to the larynx and bronchi, and is likely to cause pneumonia.

The crusts at the nasal vestibule should be softened with almond oil or a warm baking soda solution, and cleansing the nose of mucus and crusts by careful application of a wet cotton or gauze tuft is recommended. To enable the child to suck at the breast, it is given a drop of adrenalin in boric acid solution or almond oil into the nose before sucking.

Rp. Ol. Amygdalarum dulcium 10.0
Sterilis!
DS. Nasal drops

Rp. Sol. Adrenalini (1:1,000) gtt. X
Sol. Acidi borici 2% 10.0
MDS. Nasal drops

If this fails to open up the nasal passages, the child should be fed from a teaspoon. Children under three years of age should not be given cocaine and menthol.

Prophylaxis. Hardening of the body is the basic means whereby acute inflammatory diseases of the upper respiratory tract may be prevented. Its essential aim is that the body should develop a capacity for quick adaptation to changing environmental conditions, such as chilling or overheating, excess air humidity or dryness, draughts and wind, etc.

Regular exposure of the skin to open air and sunlight, cool water showers and the like, various summer and winter sports are reliable means of improving the cardiovascular and respiratory systems and ensuring normal resistance of the vascular system to unfavourable external factors.

Daily exercise of the body heat-regulating system increases body resistance to chilling and overheating by developing an intricate system of conditioned reflexes.

Exercises for hardening the whole body must be performed regularly all the year round.

In a broader sense this also implies a well-balanced diet, wearing suitable garments, as well as correct personal hygiene, both at home and at work.

Physical training and weather hardening give the body reliable protection against cold-induced ailments, as well as against many infectious and other diseases.

Gonorrheal rhinitis (*rhinitis gonorrhoica*). Gonorrheal inflammation of the nasal mucosa sometimes occurs in newborns together with gonorrheal conjunctivitis as a consequence of the child passing through the infected maternal organs. In adults, this condition is very rare, and is due to infection borne on the fingers.

Symptoms. Newborns develop marked edema and redness in the nasal mucosa on the second or third day after birth with serohemorrhagic exudation which later turns purulent.

The inflammation is very likely to extend to the external tissues of the nose and eyes and cause swelling, redness and eczematous lesions in the nasal vestibule, and conjunctivitis.

The diagnosis of gonorrheal rhinitis is confirmed by detection of gonococci in the bacteriological investigation of nasal mucus. Given appropriate treatment, the disease is of short duration.

Treatment. This is by nasal instillation of 2% protargol solution and protecting the skin of the nasal vestibule against irritation from nasal discharge.

Should gonorrhea be suspected in the mother, it is advisable to instil in the newborn's nose one drop of 1% silver nitrate solution, like it is done in eye disease prevention.

The efficacy of penicillin in the treatment of gonorrhea is indicative of its similarly powerful effect in controlling gonorrheal rhinitis.

Nasal Diphtheria
(*Rhinitis diphtherica*)

There are two types of nasal diphtheria. It is either confined to the nose or it is combined with diphtheria in the

pharynx. In the former instance, it may produce different inflammatory symptoms in the nasal mucosa and is, therefore, subdivided in clinical practice into two forms: (1) the catarrhal, or ulcero-catarrhal, and (2) the membranous.

Nasal diphtheria is the most common form of diphtheria in babies. The onset of the catarrhal form in breast-fed babies is very similar to common nasal discharge. Later, however, it becomes blood-stained, serous and purulent, with severe nasal obstruction and typical snoring. The discharge is so copious that it may excoriate the nasal vestibule and upper lip. Nasal breathing is difficult, the mouth is kept open, and a low-grade fever sets in. Nasal examination reveals crusts covering sores which bleed readily on removal of the crusts.

The membranous form of nasal diphtheria is characterised by a marked morbid coating, usually in the lower portions of both nasal cavities, which is firmly adherent to the underlying mucous membrane. The appearance of dense, whitish membranes which are difficult to peel off adds to nasal obstruction.

An attempt to remove these membranes, as well as sneezing and blowing the nose may cause free nasal bleeding.

There is no difficulty in diagnosing this condition in cases of concurrent diphtheria of the pharynx; in other cases it is made by means of rhinoscopy together with an assessment of the patient's general condition, and examination of a nasal swab for diphtheric bacteria.

The prognosis in the concomitant form of nasal diphtheria is less hopeful than in the localised form.

Treatment is by immediate administration of antidiphtheric serum, as soon as the diagnosis has been established. For treatment of the membranous type, the dose may be as large as 10,000 units. To avoid serum sickness, the injection is made preferably by Bezredka's method, whereby at first only 1 ml of the serum is injected and the full dose is given an hour later.

In the ulcero-catarrhal type, serum treatment is combined with nasal insufflation of sulfonamide powder mixed with penicillin.

Rp. Streptocidi albi
Sulfodimezini aa 2.5
Penicillini 100,000
M. f. pulv. subtilis
S. For nasal insufflation

Before insufflation, the nasal cavities should be cleansed of superfluous mucus by spraying them with adrenalin or ephedrine in 2% boric acid solution. (*Sol. Acidi borici* 2% 10.0, *Sol. Adrenalini hydrochlorici* 1 : 1,000 *gtt.* X or *Ephedrini hydrochlorici* 0.1.)

The nasal vestibule is lubricated with bland ointments to prevent dermatitis due to nasal discharge.

The bacilli carrier state and diphtheria prevention will be discussed in detail in the chapter on diphtheria of the pharynx.

CHRONIC INFLAMMATIONS OF THE NOSE (CHRONIC RHINITIS)

Chronic rhinitis occurs in three forms: (1) the so-called chronic simple rhinitis, (2) hypertrophic rhinitis, and (3) atrophic rhinitis (ozena).

Chronic rhinitis is frequently the result of repeated attacks of the common cold, i.e., acute inflammations of the nasal mucosa, or of frequent and long irritations of the nasal mucosa owing to various harmful factors. These refer to the effect of temperature, as in exposure to cold and hot air, and to harmful admixtures contained in the air inhaled, such as dust, smoke, and irritant gases. Chronic rhinitis may be caused by any of the factors liable to produce a prolonged or repeated disturbance of blood circulation in the nasal cavity, which may develop as hyperemia of the mucous membrane and its progressive congestion in certain infectious diseases, like measles, scarlet fever and diphtheria, as well as in diseases of the heart and kidneys, emphysema, obesity, etc. Chronic rhinitis may also occur by extension of an inflammation from other regions of the respiratory tract, for example, from the nasopharynx with adenoid hypertrophy, or from the paranasal sinuses, as a result of the nasal mucosa being constantly irritated by their purulent discharge.

Simple chronic catarrhal rhinitis (*rhinitis chronica simplex, s. catarrhalis*). This condition causes diffuse hyperemia and uniform swelling of the nasal mucosa. Its symptoms are basically the same, though not so marked as those of acute rhinitis. The patient, as a rule, has no constitutional disturbance. Nasal obstruction is usually worse when lying on the back or on a side. In the former instance, there is blood congestion in the lower parts of the nose, i.e., in the posterior ends of the nasal turbinates in both nasal cavities, whereas in the latter case blood congestion is confined to the turbinates of one nasal cavity. The vessels of the erectile tissue swell with blood to cause obstruction in one or both nasal cavities. When turning over in bed, the nasal obstruction will change to the dependent side. The nasal discharge is fairly liquid. Complications of chronic catarrhal rhinitis may involve full or partial loss of smell. Another frequent complication is an ear disease. The swelling of the inferior turbinate, its posterior end in particular, may directly obstruct the mouth of the auditory tube or aggravate obstructive symptoms within it, which may be followed by chronic catarrhal otitis media in consequence of prolonged partial tubal obstruction.

Finally, the lacrimal apparatus is also subject to lesions due to the swelling of the anterior end of the inferior turbinate, which may block the nearby inferior outlet of the nasolacrimal duct and cause excessive lacrimation, inflammation of the lacrimal sac and conjunctivitis.

Diagnosis. This is easily established by anterior and posterior rhinoscopy. To distinguish between the chronic simple and the hypertrophic forms of rhinitis the nasal mucosa should be painted with 0.1% adrenalin or 1-3% cocaine solutions. A nearly complete shrinkage of the nasal mucosa following cocaine application will indicate simple catarrhal rhinitis, whereas slight shrinkage or persistent swelling is characteristic of the hypertrophic form.

Prognosis. This is favourable, because swelling of the nasal mucosa can nearly always be kept down by conservative treatment or by simple surgical methods, like cauterisation with trichloracetic and chromic acids, galvanic current, etc. Relapses can be prevented with certainty only if the causes of chronic rhinitis can be eliminated.

Chronic hypertrophic rhinitis (*rhinitis chronica hypertrophica*). New growths and proliferation in the connective tissue are the most marked in the hypertrophic form of chronic rhinitis. The tissue cells usually grow for the most part in accumulated erectile tissue, rather than all over the mucous membrane, that is, at the anterior or posterior end of the inferior turbinate or at the anterior end of the middle turbinate. Sometimes, however, hypertrophy may affect the entire lower margin of the inferior turbinate. The surface of the hypertrophied areas may take on a roughened lobular or papillary ("mulberry") appearance. The posterior hypertrophied end of the turbinate sometimes forms a tumour-like protrusion into the nasopharynx. The hypertrophied areas may be pale grey-red, bright red, or purple-red in colour, depending on the amount of developed connective tissue and the degree of blood congestion. The symptoms of hypertrophic rhinitis are similar to those of simple rhinitis. Nasal obstruction which results from more permanent factors, such as hypertrophy of the mucosa, is more persistent, scarcely diminishes even after the application of vasoconstrictive drugs, and unlike simple rhinitis is not prone to change with changes of the head and body position. The mucous secretion is thicker and sticky. Violent blowing of the nose through both nostrils often leads to a middle ear disease.

Prognosis. This is comparatively good due to the possibilities of removing some of the hypertrophied areas of the nasal mucosa and thus re-establishing the nasal airway.

Treatment. First, it is necessary to remove all the factors that produce or sustain the condition. Therefore, considerable attention must be paid to the patient's general condition and establishing whether or not he suffers from heart or kidney disease, obesity, etc., as well as to learn about the sanitary and other relevant conditions of his occupation. After these conditions have been ascertained and improved, there is a distinct likelihood that local conservative treatment as well as surgical may be effective. The conservative treatment of simple chronic rhinitis is in many cases limited to the use of astringents or caustics. Among the former, 1%-2%-3% protargol solution for nasal drops is commonly used. Vasoconstrictive drugs, such as ephedrine, may also be used in the following formula:

Rp. Sol. Ephedrini 3% 10.0
D.S. Dose of five drops to be repeated twice daily

A more usual method of shrinking swollen nasal mucosa is to paint it with 0.25-0.5% iodine glycerol solution. Measures against hypertrophic rhinitis should be more vigorous,

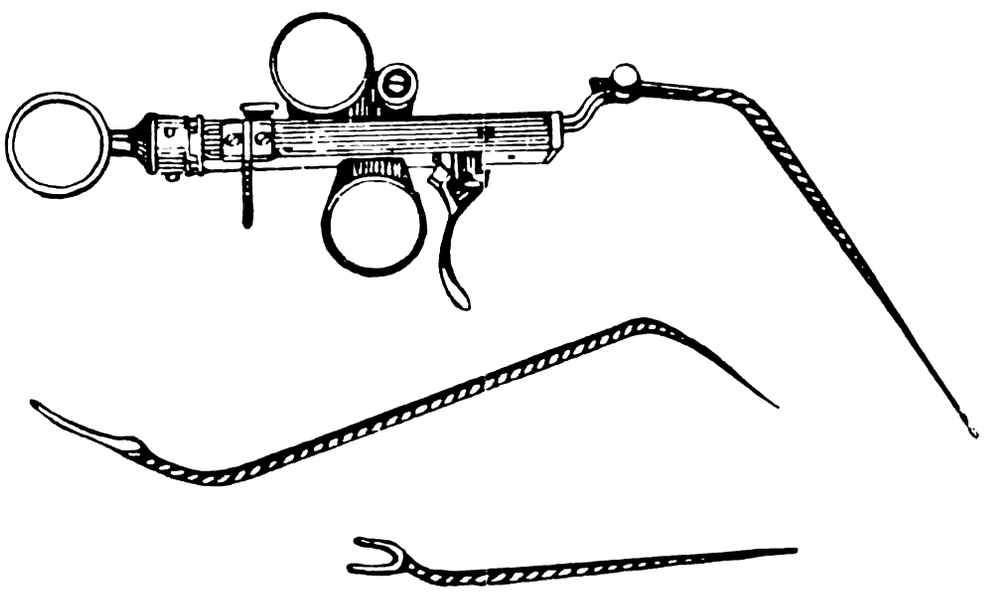


Fig. 53. Galvanocauter with Handle and Detachable Needle Pieces

such as cautery of a diffuse swelling in the presence of mild hyperplasia of the mucous membrane. This is done either with chemicals, like chromic and trichloracetic acids or a silver nitrate stick, or else with a galvanocauter (Figs. 53-54).

In chromic acid cauterisation, a bead of acid is fused to the thick probe end by heating it to melting point in the flame of an alcohol lamp. Overheating should be avoided, for this will turn the acid brown and its caustic properties will be lost.

It is sufficient to draw one or two lines from behind forward along the margin of the turbinate with the acid-tipped probe or a pointed cauter, to produce con-

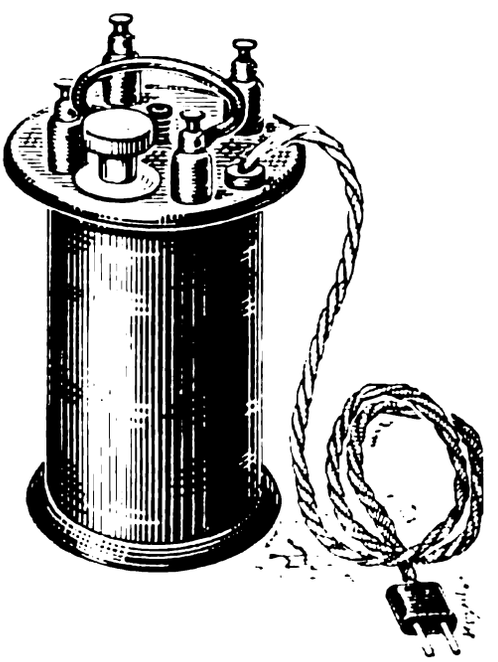


Fig. 54. Rheostat for Galvanocautery

traction of a swollen or hypertrophied mucosa (Fig. 55) around the scar when the slough comes away, with the accompanying restoration of the erectile tissue to normalcy.

When necessary, a stronger coagulative effect followed by a greater contraction may be produced by drawing one or two lines along the turbinate margin with a pointed electrocauter.

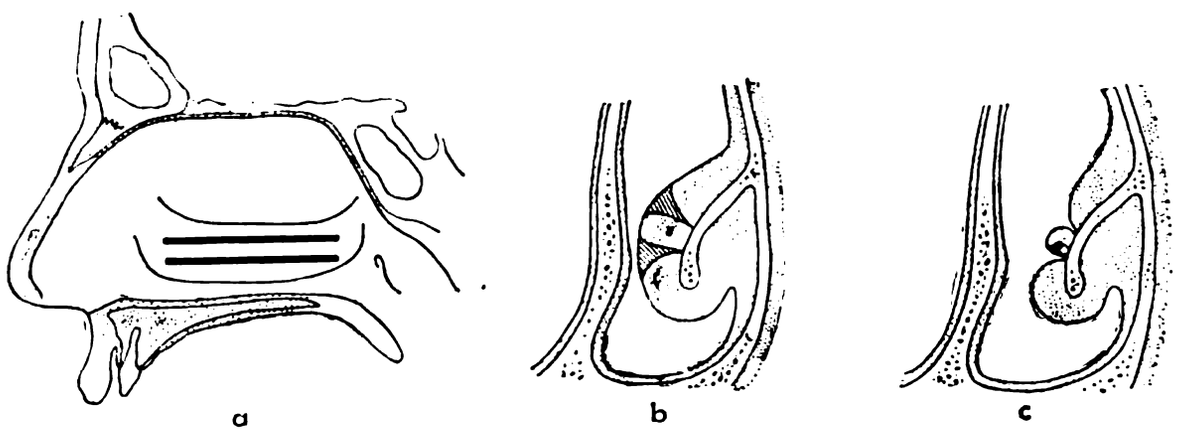


Fig. 55. Galvanocautery of Turbinates

(a) galvanocautery of turbinates. Lines show places of cauterisation on inferior turbinate; (b) nasal turbinate before cauterisation; (c) nasal turbinate after cauterisation

When removing isolated and limited hypertrophied areas in the nasal mucosa, which are usually confined to places of marked erectile tissue accumulation, preference should be given to the use of the nasal snare (Fig. 56) and nasal scissors (Fig. 57).

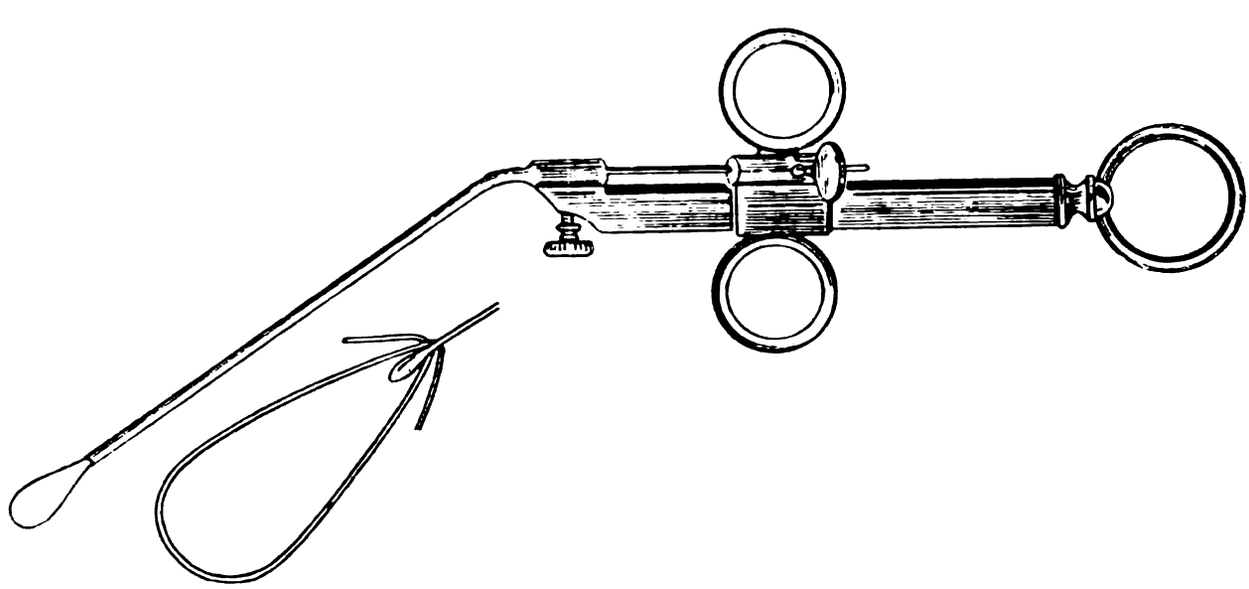


Fig. 56. Nasal Snare

Atrophic rhinitis and ozena (*rhinitis atrophica simplex et ozaena*). Simple atrophic rhinitis is a chronic disease of the nasal cavity, characterised by atrophy of the mucosa which grows thin and loses some of its mucous glands. The disease is often accompanied by diminished mucous secretion which tends to dry into crusts, but has no fetor.

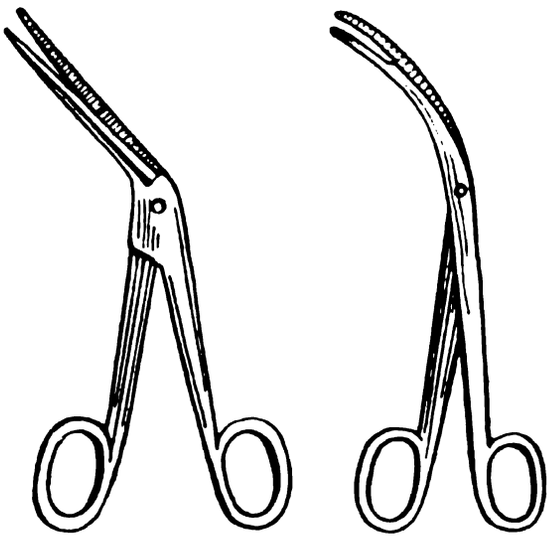


Fig. 57. Nasal Scissors for Removing Inferior and Middle Turbinates

Many theories have been advanced to explain the cause of atrophic rhinitis, but none of them can account for all of the clinical symptoms seen in this disease. It may be assumed that both atrophic and hypertrophic changes occurring in the nose are due to a single process of trophic disturbances in the nasal tissue. Some theorists, therefore, hold the view that atrophic rhinitis always develops from hypertrophic rhinitis of which it is simply a later stage. It is true, no one has directly observed the transition of hyperplasia to atrophy, but their relation is considered possible in view of their frequent joint appearance. The assertion of formal geneticists to the effect that this disease is congenital holds no water, as no scientific proof exists to support their view.

Numerous investigations of Soviet scientists have established that atrophic rhinitis may be due to environmental causes, such as dust and unfavourable climate. The effect of silicate, cement, tobacco and some other kinds of dust is especially pernicious.

Subjective symptoms. These are not always very marked. The patient complains in the main of constant dryness in the nose and nasopharynx. Another common complaint is one of a feeling of dull pressure at the nasal root, and headache. There is severe nasal obstruction owing to heavy crusting. The decay of the crusts will lend them a fetid odour. This condition is known as *ozena*.

The basic signs of ozena are the following: (1) heavy crusting in the nose; (2) a peculiar fetor; (3) severe atrophy of the nasal mucosa as well as of the bony structure of the turbinates.

In addition, ozena is commonly accompanied by total anosmia owing to the spread of atrophy to the olfactory area. After removal of the crusts, the nasal passages are seen to be very wide and in anterior rhinoscopy offer a clear view of the posterior wall of the nasopharynx. The crusts cause severe itching, and the patient is constantly tempted to pick the nose, which frequently leads to injury and subsequent perforation of the nasal septum.

Etiology. The true cause of ozena is unknown. Many theories have been offered to explain it, none of them reliable. Neither is there any valid proof that this disease is communicable, although the fetid odour from the nose makes the patient a social outcast.

No doubt, environmental factors, of social nature in particular, have a bearing on the origin and course of ozena. In the past quarter-century, its incidence has shown a marked decrease owing to the rising welfare and cultural standards of the mass of the Soviet people.

Diagnosis. This is easily established by rhinoscopic examination. Other signs aiding diagnosis are characteristic complaints, marked atrophy of the nasal mucosa, and foul-smelling crusts with anosmia commonly present. Ozena, however, produces no ulceration in the nose, as distinct from nasal tuberculosis and syphilis.

Prognosis. Reliable prognostication in ozena is doubtful. True, it is not difficult to relieve the most troublesome symptoms, but efforts to restore the nasal mucosa to full normal function have hitherto been unsuccessful. This also refers to the sense of smell.

Treatment. In ozena, just as in simple atrophic rhinitis, the treatment may only be symptomatic. It aims to relieve dryness and crusting in the nose by alkaline douches with subsequent application of iodine glycerol solution, ointments or menthol oil. The nose may also be cleansed of crusts by a warm alkaline spray.

Below are the formulae of the alkaline solution and some of the drops and ointments commonly used:

Rp. Jodi puri 0.05-0.1
Kalii jodati 0.2
Glycerini
Aq. destill. aa 5.0
Ol. Menthae pip. gtt. 1
DS. Five drops in each nostril twice a day

Rp. Mentholi crystallisati 0.1
Ol. Provincialis 20.0
DS. Five drops in each nostril twice a day

Rp. Natrii bicarbonici 60.0
Natrii biborici 30.0
Natrii chlorati 10.0
Ol. Menthae pip. gtt. V.
M. f. pulv.
DS. One teaspoonful in a glass of water for a nasal douche

In view of the fact that ozena is accompanied by the growth of innumerable bacteria in the nasal cavity, which leads to a characteristic fetid odour, a combination of antibiotics and nicotinic acid has been suggested for treatment of ozena. This treatment should be given only if prescribed by a specialist. Biomycin (auromycin, terramycin) is given day and night once in every six hours in an overall dose of 1.2 g. At the same time, nicotinic acid is given orally in 50 mg doses three times a day and only once daily by a 50 mg intramuscular injection. The duration of treatment is between 10 and 14 days. Streptomycin may be given instead of biomycin in a 0.5 g dose by intramuscular injection repeated twice daily. In addition it is helpful to spray the nasal cavities every day with streptomycin solution containing 0.5 g of the drug per 10 m of physiological solution. This treatment may be repeated in a few months' time.

VASOMOTOR OR ALLERGIC RHINITIS
(*Rhinitis vasomotorica*)

Vasomotor rhinitis is characterised by intermittent attacks with very brief and sometimes long periods of relief. The attacks, very violent at times, are accompanied by prolonged paroxysms of sneezing, nasal obstruction and profuse, mostly watery, discharge from the nose. Many patients have additional symptoms of lacrimation, itching of the

eyes, nasal interior and hard palate, and headache. The nasal mucosa is edematous and swollen, the meatuses are filled with a watery or foamy secretion, and nasal breathing is highly inadequate. In the intervals between the attacks, all morbid symptoms may utterly disappear, but in protracted cases, the swelling of the nasal mucosa may become stable or turn into hyperplasia, as in chronic rhinitis. Vasomotor rhinitis mostly occurs in subjects with a hypersensitive nervous system. It is mainly caused by abnormal reactions of the body, as a result of which a number of external and internal factors, such as temperature and atmospheric pressure changes, the effect of chemicals, emotional strain, etc., may give rise to inadequate, physiologically groundless body reactions. Even a slight chilling or some other irritation of the central or peripheral nervous system may cause a sudden and acute attack of rhinitis in such subjects, which very often passes just as suddenly.

In view of a changed body reactivity, vasomotor rhinitis frequently occurs together with bronchial asthma, angioneurotic edema, nettle rash, etc. Some forms of vasomotor rhinitis, therefore, can be regarded as a peculiar local anaphylactic reaction to an unknown allergen. Cytological examination of swabs taken from the mucous membrane of the inferior turbinates reveals that many patients have marked eosinophilia, i.e., eosinophils accounting for more than half of the aggregate number of blood cells.

Treatment. Measures should be taken to normalise the function of the central nervous system first and foremost. The heightened body reactivity is reduced by appropriate hardening of the whole organism which implies wearing rational garments, getting plenty of fresh air, and other hygienic habits. The following formulae of bromine and caffeine drugs are recommended for treatment of the central nervous system:

Rp. Natrii bromati 2.0
Coffeini natrio-benzoici 0.5
Aq. destill. 200.0
MDS. One tablespoonful three times daily

Physiotherapeutically, ionisation of cervical sympathetic nodes with the use of calcium chloride may be prescribed.

So far as medicines are concerned, calcium, atropine, histamine, and vitamin C preparations are used apart from drugs that serve to heighten general body resistance and tone. In certain cases, there is marked improvement following treatment with dimedrol given in single doses of 0.05 g two to three times daily for a period of 10 to 12 days. Another measure sometimes helpful is novocaine blocking by injection of 2 ml of 1% novocaine solution into the mucous membrane of the posterior end of the inferior turbinate. The troublesome condition associated with swelling of the nasal mucosa and difficult nasal breathing is partly relieved by instillation into the nose of the following drops: *Cocaini muriatici*, *Ephedrini hydrochlorici aa* 0.2, *Sol. Acidi borici* 3% 10.0.

In the absence of contraindications, considerable relief may sometimes be afforded by adrenocorticotropic hormones (ACTH) and cortisone, which bring down exudation.

Nasal anatomical deformities such as deviations, spurs and ridges of the nasal septum, and hypertrophy of the turbinates, which impede nasal breathing or irritate the nasal mucosa, should be corrected by surgery.

Hay fever. As a specific variety of vasomotor rhinitis, hay fever is an exclusively allergic syndrome. It is a seasonal disease occurring during pollination of plants, when inhalation of air with an admixture of pollen, sometimes a quite insignificant amount, is enough to produce this condition in people hypersensitive to this allergen. In stubborn cases, the attacks of rhinitis are so exhausting that the patient has to move to another climate at the appropriate season, preferably to a sea-shore with scanty vegetation.

DISTURBANCES OF THE SENSE OF SMELL

Impairment of the sense of smell, otherwise called hyposmia, occurs in different degrees in all acute and chronic inflammations of the nasal mucosa, as well as in all anatomical deformities obstructing nasal breathing, and may be transient or permanent.

Severe anatomical deformities in the nose which prevent the inhaled air containing odoriferous particles from entering the olfactory area may cause full loss of the sense of smell,

known as anosmia. This condition is called *respiratory* anosmia.

In some cases, anosmia is due to destruction of the receptors of the olfactory analysor, namely, its nerve endings and olfactory cells. This destruction mostly occurs by extension of the inflammatory process to the olfactory area, as, for instance, in ozena, chronic suppurations in the posterior paranasal sinuses, and so on. There have been cases of lesions of the olfactory nerve itself (neuritis) in intoxications by various chemicals, like nicotine, morphine, and atropine, or following infectious diseases, such as influenza and diphtheria. Total loss of smell due to olfactory nerve affection is called *essential* anosmia. Anosmia is sometimes produced by a pathological condition in the brain, such as a cerebral abscess, tumour or encephalitis, known as *central* anosmia.

Prognosis. In respiratory anosmia, the prognosis is favourable, since the olfactory sense may be restored by removing the mechanical obstacle to respiration. In essential anosmia, the forecast is usually hopeless, as the dead nerve endings cannot be vitalised. In central anosmia, the prognosis depends on the nature of the basic disorder and is often unfavourable.

Treatment. Treatment should be directed towards the removal of obstacles leading to the olfactory fissure. In essential and central anosmia the basic requirement is to cure the principal disease.

NEOPLASMS IN THE NOSE

Benign new growths. Benign nasal growths rarely met with are *papilloma*, which for its rough surface resembles a cauliflower, as well as *angioma*, *fibroma*, *chondroma* and *osteoma*.

The most frequent growth is the nasal *polyp*.

In origin and histological structure, nasal polyps are the products of a chronic inflammation of the mucous membrane. For this reason, it is not quite correct to classify them among neoplasms, although this is done owing to their superficial likeness to the latter and their tendency to quick growth and relapse after removal.

Nasal polyps are single or multiple masses attached

by a pedicle that mostly arise from the middle nasal meatus. They are greyish or, sometimes, yellowish-red in colour, with a smooth surface and are of jelly-like consistency. Their size and number vary within broad limits. In some cases, the nasal cavity is filled with small polyps, but usually one polyp grows so large that it fills the entire nasal cavity and even projects outside through one of the anterior nares. In other cases, a polyp may grow backwards, towards the choanae. On passing through a choana, this type of polyp, which is termed the choanal, may fill the entire nasopharynx.

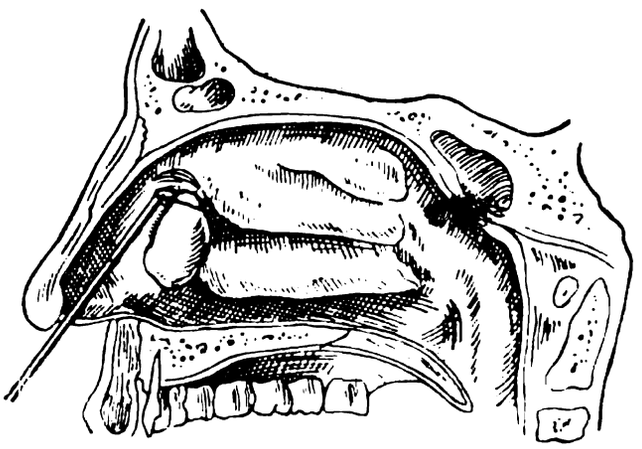


Fig. 58. Removal of Nasal Polyp with Wire Snare

Polyps are caused by a chronic inflammation of the mucous membranes of paranasal sinuses, commonly the antrum of Highmore and the cells of the ethmoid labyrinth.

Histologically, the polyp is a delicate fibrous mass of reticular connective tissue containing numerous plasma cells and lymphocytes as well as spaces filled with serous fluid.

Diagnosis. Nasal polyps are readily identified owing to their characteristic symptoms and appearance.

Treatment. The only cure for nasal polyps is their surgical removal with a nasal snare (Fig. 58) under local anesthesia. The nasal mucosa is painted with 5-10% cocaine solution with adrenalin. The wire loop of the snare is threaded over the polyp and moved towards the pedicle base, whereupon the polyp is avulsed with a gentle clossure of the snare.

Malignant neoplasms. Malignant new growths found in the nasal cavity are due to *carcinoma* and *sarcoma.* They may originate from any one of the walls of the nasal cavity, but they usually extend from the maxilla and ethmoid labyrinth. Among the wide variety of symptoms characteristic of malignant new growths in the nose, the basic are those of

unilateral nasal obstruction and, frequently, of nasal bleeding. A further growth and decay of the tumour will produce a foul, purulent discharge. When the tumour invades the surrounding tissues, the eyeball is displaced to the degree of bulging, eyesight is impaired or totally lost, and there may be intracranial complications. A rapid growth of the tumour is very likely to produce bulges on the cheeks and the palate.

The tumour is mostly of cauliflower appearance and bleeds readily when touched with a probe. It is set on a broad base and cannot be avoided in probing.

An accurate diagnosis is based on the clinical record, X-ray examination of the skull and histological investigation of a biopsy of the tumour.

Prognosis. In malignant new growths of the nose the prospect is well nigh hopeless, particularly where the onset of the condition has been overlooked.

Treatment. The common procedure is radiotherapy combined with surgical interference.

ACUTE AND CHRONIC DISEASES OF PARANASAL SINUSES

Acute maxillary sinusitis (*sinuitis acuta maxillaris*). Acute inflammations of the antrum of Highmore frequently occur with acute rhinitis, influenza, measles, scarlet fever and other infectious diseases, as well as following an injury to the paranasal sinus.

In mild cases of this disease, the subjective symptoms are those of a feeling of pressure and tension in the region of the affected sinus and unilateral nasal obstruction. In more severe cases, in addition to these symptoms there will be pain, frequently confined to the region of the forehead and zygomatic bone, and toothache. This is followed by a swelling of the cheek on the affected side, the onset of fever and malaise. Anterior rhinoscopy usually reveals congestion and swelling of the mucosa of the middle nasal meatus containing excessive mucous secretion and, sometimes, pus which runs in a string from under the middle turbinate.

Since acute inflammation of the mucosa or pus in the middle nasal meatus may with equal reason be ascribed either to lesion of the maxillary and frontal sinuses or to

lesion of the ethmoid cells, the final diagnosis rests on additional information gained by transillumination, skiagraphy, proof puncture and irrigation of the antrum of Highmore.

Transillumination of the paranasal sinuses, otherwise known as diaphanoscopy, is made in complete darkness with a bright electric light placed within the patient's mouth (Fig. 59). When the lips are closed, a cherry-red glow will show through both sides of the face. A dimmer glow on any facial side may provide evidence of lesion of the maxillary or ethmoid sinus. The degree of pneumatisation of the frontal sinuses is determined by placing the lamp against the inner corner of the orbit, i. e., the floor of the frontal sinus.

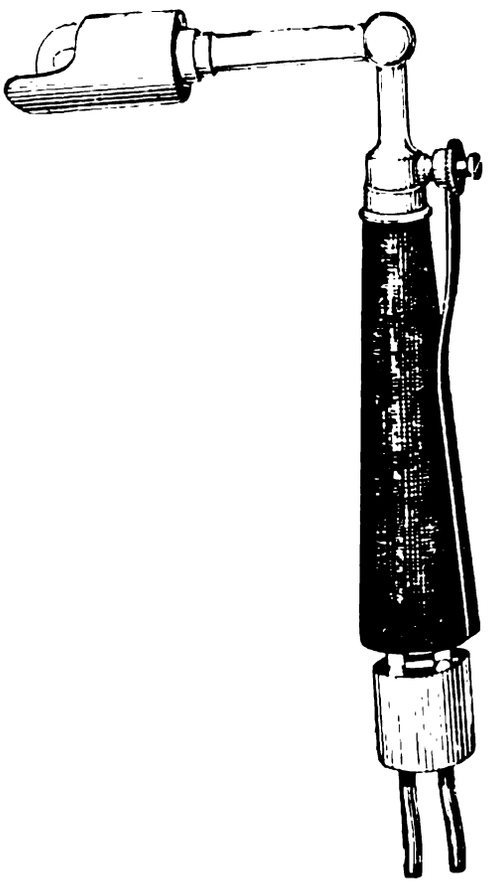


Fig. 59. Transilluminating Lamp for Paranasal Sinuses

Examination of the middle nasal meatus may be facilitated by cocainisation and, sometimes, by probing the natural orifice of the maxillary sinus proper.

The puncture of the antrum of Highmore on the side of the middle meatus is made by means of a special cannula or needle (Fig. 60). The antrum is usually punctured through the inferior meatus with a 6 to 8 cm long thick straight needle or trocar (Fig. 61). Following the puncture, an attempt should be made to aspirate pus with a syringe, following which the sinus is washed out with 4% boric acid solution.

The condition of the antrum is ascertained by the quality of the liquid used.

Treatment. In cases of acute inflammation of the paranasal sinuses with a high-grade fever, the patient should be kept in bed and treated with febrifuges, such as aspirin (acetylsalicylic acid) and caffeine. These are given in 0.5 g and 0.1 g powder doses respectively two or three times daily. To reduce swelling of the mucosa in the natural ori-

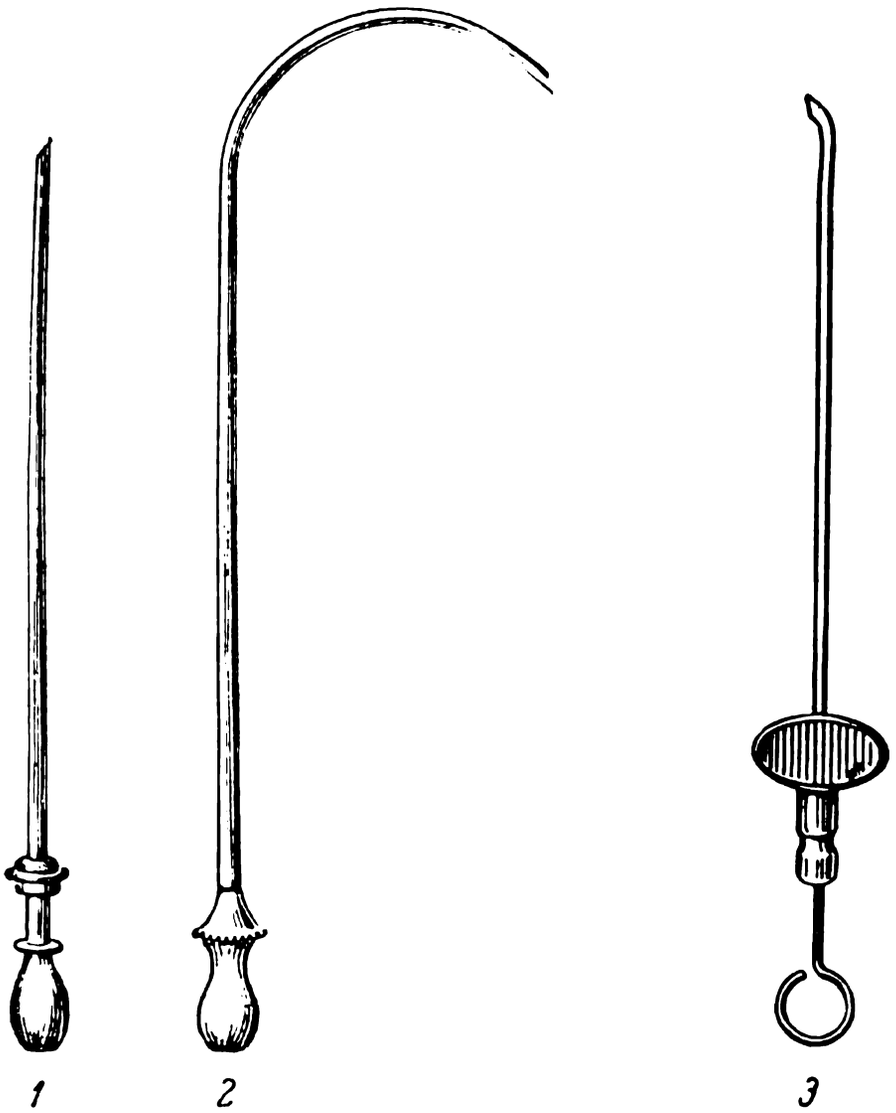


Fig. 60. Needles for Puncture of Antrum of Highmore
(1) trocar for spinal puncture; (2) Stein's needle; (3) Kulikovsky's needle

fices of the paranasal sinuses and to promote the drainage of secretion, a 5% solution of cocaine is used to paint the middle nasal meatus, as well as the instillation of cocaine drops with adrenalin into the nose several times daily.

Rp. Cocaini hydrochlorici 0.2
Sol. Adrenalini 1:1,000 gtt. X
Sol. Acidi borici 4% 10.0
DS. Five drops in each nostril three to four times a day

Ephedrine drops are used for the same purpose.

Rp. Sol. Ephedrini hydrochlorici 3% 10.0
DS. Five drops in each nostril three to four times daily

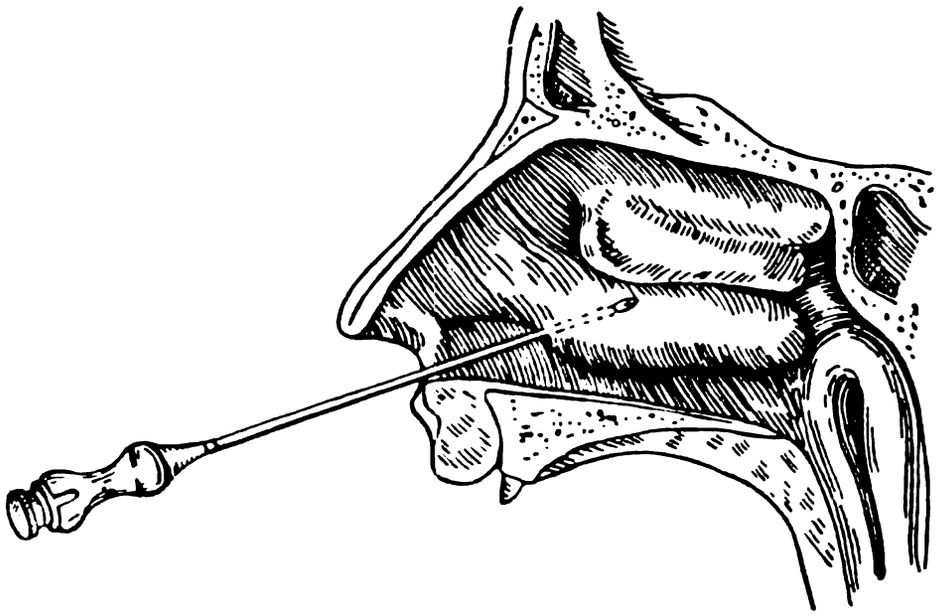


Fig. 61. Puncture of Antrum of Highmore through Inferior Nasal Meatus

The patient should be taught the correct technique of drop instillation in which the head should be tilted far back and a little towards the affected side to let the drops get into the middle nasal meatus.

The natural orifice of the paranasal sinus narrowed by the swollen mucosa broadens under the effect of treatment, and in certain cases pus may flow directly out of the middle nasal meatus.

An effective cure in acute maxillary sinusitis is a hot compress, blue, "sollux" and ultraviolet light, as well as ultrashort-wave diathermy, which is given in daily quarter-hour sessions for 12 to 15 days and is particularly helpful. If, ten to twelve days following the onset of this condition its acute symptoms have subsided, and sinus drainage is free, diathermy may be used to cure residual symptoms, as well as puncture of the antrum followed by its injection with penicillin and streptomycin.

Chronic maxillary sinusitis (*sinuitis chronica maxillaris*). Although chronic inflammation of the antral mucosa usually results from residual symptoms of an acute inflammation, it may arise in the absence of a preceding acute process by extension of a chronic inflammation from the nasal

cavity or other paranasal sinuses, above all the ethmoid labyrinth. Another cause of chronic highmoritis may be an inflammation in the root of a tooth.

Symptoms. The subjective symptoms are unmarked. The patient complains of unilateral nasal obstruction, more or less copious nasal discharge, often purulent, headache, and early weariness from mental exertion. A fairly common symptom is hyposmia and, sometimes, perception of a foul odour in the nose, known as cacosmia. Physical examination reveals pathological changes in the middle nasal meatus, such as inflammatory symptoms in the mucosa and polyps, as well as a purulent or mucopurulent discharge which quickly reappears after being wiped off with a cotton tampon.

Diagnosis. The diagnosis of chronic maxillary sinusitis is based on the record of complaints and physical signs. A differential diagnosis is facilitated by auxiliary methods, such as X-ray photography, transillumination, proof puncture and irrigation of the antrum of Highmore.

Treatment. An effective conservative treatment of chronic maxillary sinusitis is by irrigation of the antrum with a saline solution followed by

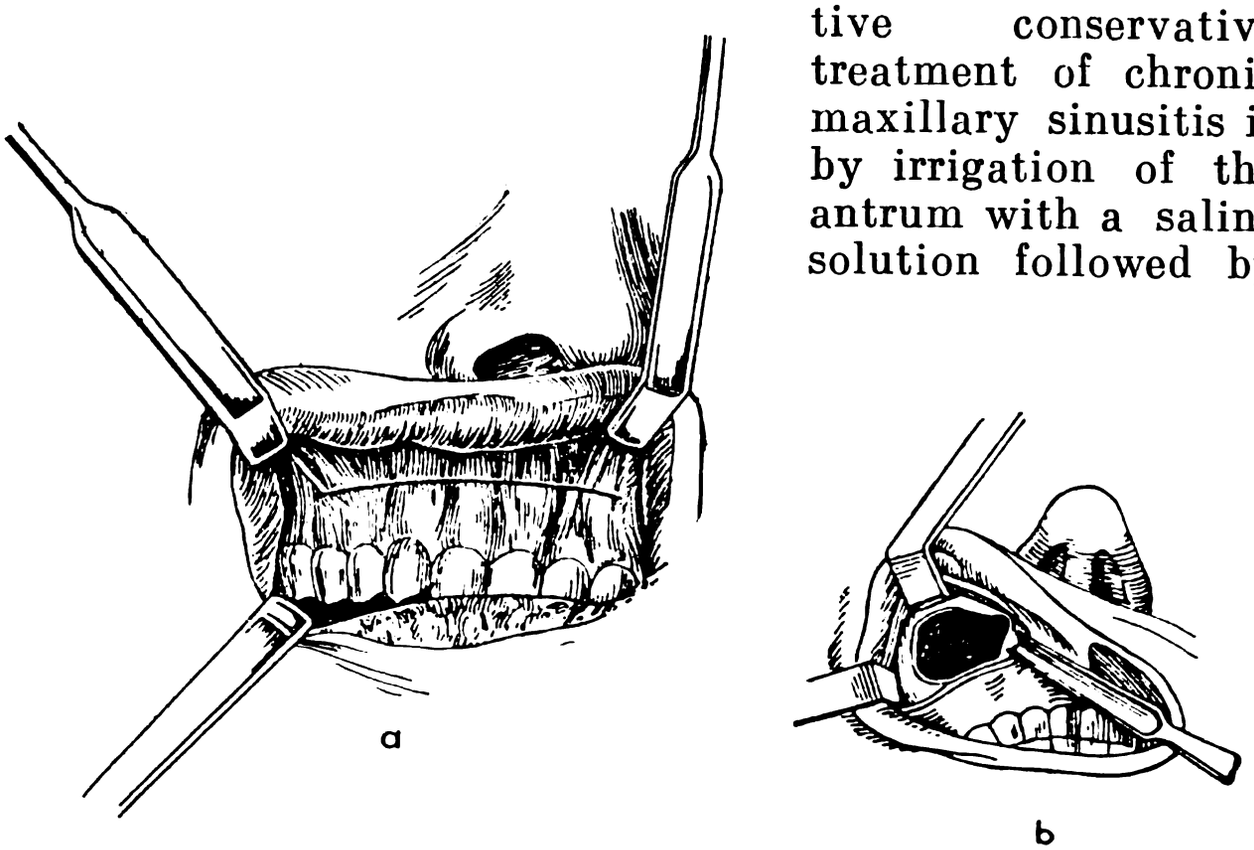


Fig. 62. Radical Operation on Maxillary Sinus
(a) incision; (b) opening of anterior bony wall

its injection with penicillin or streptomycin solutions, 200,000 to 250,000 units in a 2 or 3 ml dose.

The irrigation is made with the aid of a puncture needle following puncture of the antrum in the inferior nasal meatus. In light cases, diathermy, ultra-high frequency currents, and "sollux" light therapy may sometimes prove successful.

In the majority of cases, however, surgery is indicated for the removal of polyps and granulations from the sinus, and to provide for a wide communication between the latter and the nasal cavity. An improved technique of this operation has been suggested by A. F. Ivanov.

The first stage is incision of the mucosa under the upper lip (Fig. 62), which elevates the soft tissues to expose the anterior bony wall of the antrum. The wall is opened as wide as possible with a chisel and forceps (Fig. 63). The initial cut is always made at the site of attachment of the maxillary zygomatic process, as this will always provide access to the sinus interior, however small it may be. Pus and diseased mucosa are then removed from the sinus through the opening in the anterior antral wall, whereas normal mucosa is left in situ. Communication with the nasal cavity is set up by removing part of the internal wall of the antrum in the inferior nasal meatus (Fig. 64). The operation is performed under local anesthesia.

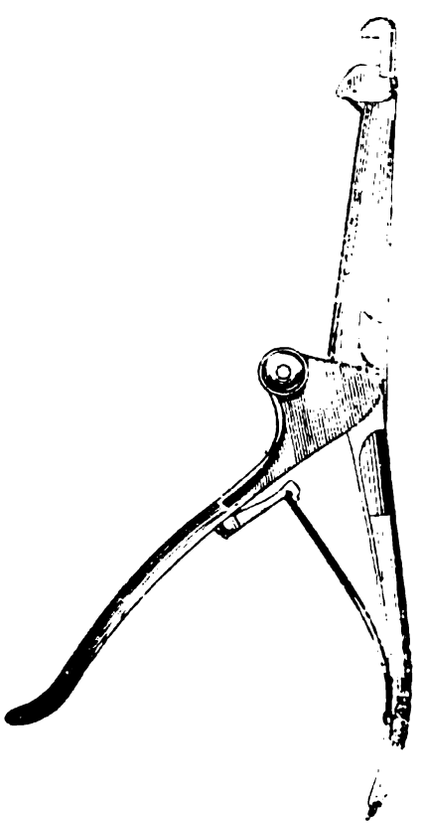


Fig. 63. Bone Forceps for Removal of Anterior Wall of Maxillary Sinus

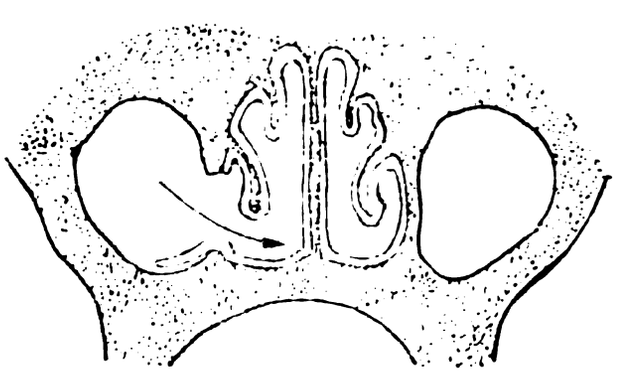


Fig. 64. Newly-Formed Window between Antrum and Inferior Nasal Meatus in Radical Operation on Maxillary Sinus

Following the operation, an ice-bag is applied to the cheek to keep down the swelling. The patient is given cold liquid food for three to four days. Postoperative treatment consists of antiseptic douches through the newly-formed naso-antral window given four to five days after the operation.

Prophylaxis. Prevention of inflammatory diseases of the maxillary sinuses is by a correct and timely treatment of both acute and chronic rhinitis as well as by treatment of the upper molar teeth which have a direct relation to these sinuses.

Acute frontal sinusitis (*sinuitis acuta frontalis*). The circumstances and causes of frontal sinusitis are the same as in maxillary sinusitis. The basic complaint in this condition is one of pain and exquisite tenderness on finger pressure over the floor of the frontal sinus at the inner corner of the orbital roof. Frontal sinusitis may be accompanied by photophobia, lacrimation and pain in the orbit. In severe cases, particularly with poor drainage from the frontal sinus, there may be swelling in the upper eyelid and over the brow.

Anterior rhinoscopy will reveal the same physical signs as in maxillary sinusitis.

Diagnosis. This is made with the aid of transillumination and X-ray films.

Treatment. The most common remedy is conservative treatment. Penicillin is given by intramuscular injection four to six times a day to allay fever and headache and promote recovery.

Furthermore, the drainage of secretion from the sinus must be ensured by painting the middle nasal meatus with cocaine, and sometimes a little surgery is required in this area to remove the anterior end of the middle turbinate.

Various physiotherapeutic procedures, including exposure to blue and "sollux" light, as well as ultrashort-wave diathermy are most helpful.

Chronic frontal sinusitis (*sinuitis chronica frontalis*). The subjective symptoms of this disease are much milder than in acute inflammation of the same sinus. These symptoms may be aggravated, however, by poor drainage of the frontal sinus with a resulting pressure increase within it and ex-

treme tenderness on pressure over its floor. The pain in the frontal sinus may increase due to the inflow of blood to the head precipitated by strong drink, smoking, night-work, etc. A fistula may sometimes develop in the inner corner of the orbit.

Diagnosis. This is usually easily established after an analysis of all subjective symptoms and physical signs has been made following auxiliary diagnostic procedures, like transillumination, X-ray photography, and probing the nasofrontal duct.

Treatment. Surgical interference is indicated should conservative measures fail to cure the grave forms of frontal sinusitis with a headache so severe as to render the patient incapable of working. Clear indications for a radical operation on the frontal sinus are also lesions of the bony walls, fistulas and, above all, complications occurring by extension of the inflammatory process to the tissues within the cranial cavity or the orbit.

The operation is, as a rule, performed under local anesthesia. The incision of the skin and periosteum is carried along the brow, past the inner corner of the eye, and down along the nasal bone to the lower ridge of the orbit (Fig. 65). The soft parts are pushed upwards and downwards to expose the anterior wall and floor of the frontal sinus, whereupon a wide hole is made in the sinus floor. Morbid mucosa, pus and granulations are then removed from the sinus, and a wide passage is formed between the frontal sinus and the nasal cavity.

In view of the fact that the anterior ethmoid cells are usually affected together with the frontal sinus, A. F. Ivanov has suggested that the so-called *fronto-ethmoid* trephination be performed in all cases of the above-described operation by a simultaneous opening of the ethmoid cells. The operation ends with insertion into the frontal sinus of a thick-walled rubber drain passed through the nose and the naso-frontal passage made during the operation. The incision is securely closed with stitches in the soft tissue.

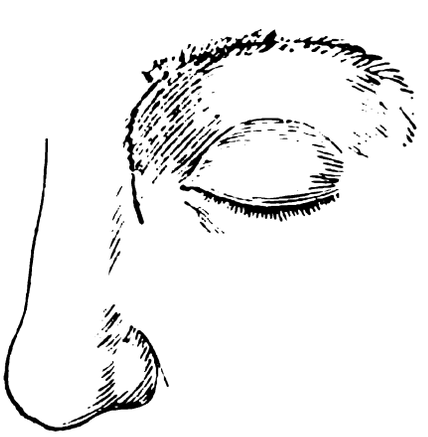


Fig. 65. Skin Incision in Radical Operation on Frontal Sinus

Postoperative treatment is similar to that employed in the opening of the maxillary sinus, that is, by periodical antiseptic douches of the frontal sinus.

Acute and chronic ethmoiditis (*sinuitis ethmoidalis acuta et chronica*). The symptoms of acute inflammation of the ethmoid cells resemble those of frontal sinusitis.

The fact that pain is mostly felt at the nasal root and the inner corner of the orbit may indicate affection of the ethmoid cells. In chronic cases, subjective symptoms may with equal reason be associated with either frontal or ethmoid sinus disease, yet there may also be an absence of symptoms. Rhinoscopy in chronic forms of ethmoidal inflammation will reveal more or less typical signs. Frequently the middle nasal meatus is filled with polyps with pus oozing between them. The presence of pus in the superior nasal meatus above the middle turbinate is diagnostic of suppuration in the posterior ethmoid cells or the sphenoid sinus.

Treatment. In *acute cases*, the treatment is the same as in acute frontal sinusitis.

In chronic cases, surgery is required to open all of the affected ethmoid cells. This operation is usually performed via the nose with a special set of instruments (Fig. 66, a). After the cells have been opened, tags of torn tissue are removed with a conchotome or forceps (Fig. 66, b) and with a nasal snare, the entire space of the ethmoid labyrinth being cleansed.

In the event of a lesion of the anterior cells adjoining the floor of the frontal sinus and in the presence of external fistulas, this operation is made through an external incision on the face, as in cases affecting the frontal sinus.

Sphenoiditis (*sinuitis sphenoidalis*). Sphenoid sinus suppuration is commonly connected with a lesion of the posterior ethmoid cells which closely adhere to the sphenoid sinus. A distinctive symptom of this condition is remittent headache with reflex pain in the nape, or less frequently in the forehead or vertex. Rhinoscopy will show pus in the superior nasal meatus which flows down into the nasopharynx. Probing and irrigation of the sphenoid sinus both form part of the treatment and diagnosis. Intranasal surgical opening of the sphenoid sinus is sometimes indicated.

Orbital and intracranial complications. An acute or chronic inflammation in the paranasal sinus may give rise to

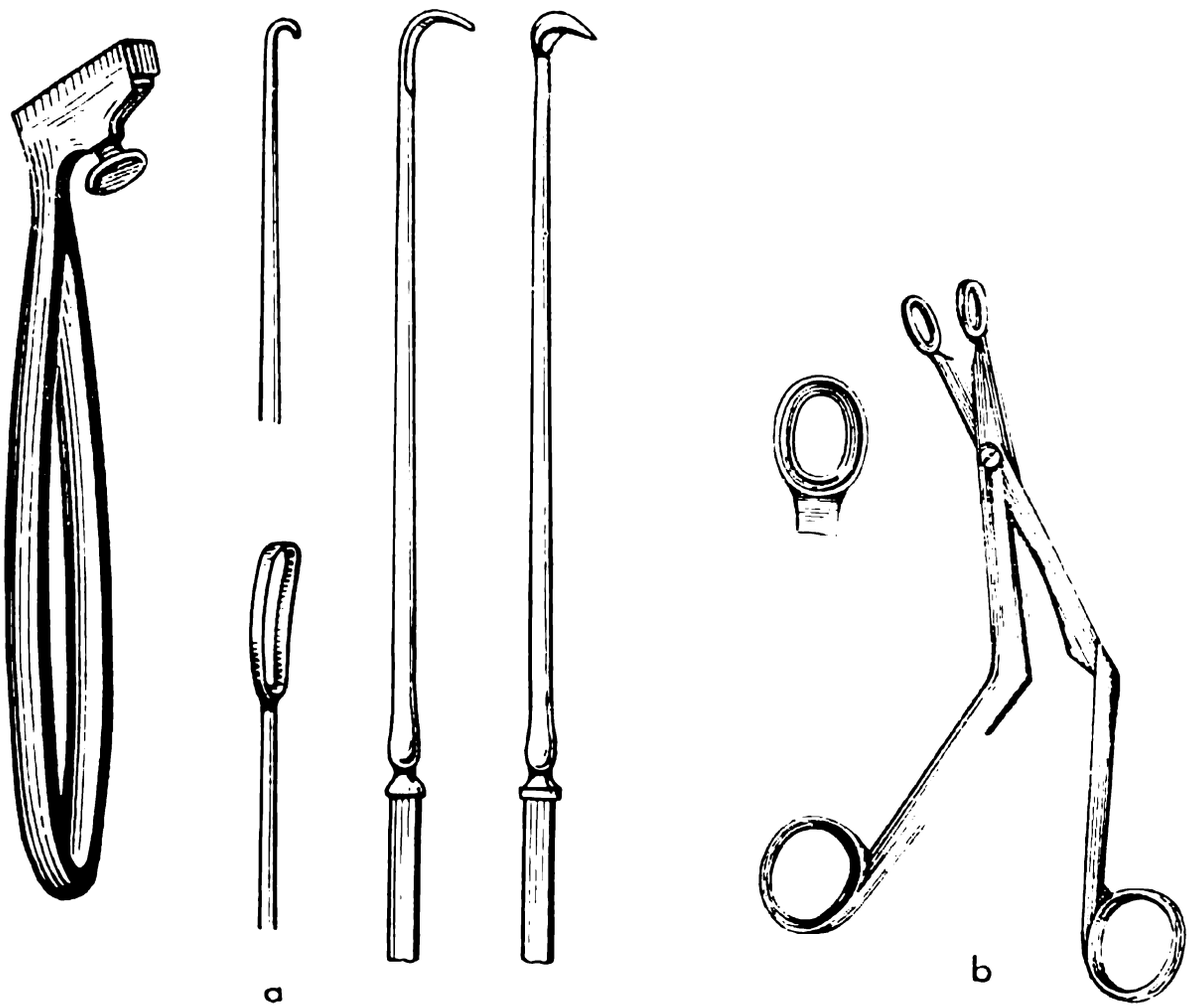


Fig. 66.

(a) instruments used in operations on ethmoid labyrinth; (b) nasal forceps

intraorbital and intracranial complications, which are sometimes fatal. These complications may arise by direct contact between the infected tissue and the contents of the orbital or cranial cavities as well as by extension of the infection by blood and lymph vessels. The veins which directly connect the superior part of the nasal cavity and the paranasal sinuses with the longitudinal sinus of the dura mater may provide a route for the infection extending into the latter sinus and causing its phlebitis.

The pressure of the inflammatory infiltrate on the eyeball and ophthalmic nerve may markedly displace the eyeball or cause it to protrude. Another result is loss of vision which in mild cases may be quickly restored, as soon as the basic cause of the condition is eliminated.

In some cases of closed empyema within the ethmoid cells, the infection may spread to and cause suppuration in the postocular cellular tissue with a danger of complications threatening the organ of sight and the life of the patient.

The antrum of Highmore is more frequently the source of orbital rather than intracranial complications.

Among intracranial complications, such as an extradural abscess, purulent meningitis and cerebral abscess, the latter is the most dangerous and often proves fatal.

Timely treatment of inflammations in the paranasal sinuses will prevent these dangerous complications. In the event of suspected intracranial complication a radical operation will be required followed by intensive antibiotic therapy with large doses of penicillin, streptomycin, and the like.

Should the condition be serious enough to warrant surgical interference in the cranial cavity, the common rules of brain surgery must be employed.

DISEASES OF THE PHARYNX

ANATOMY OF THE PHARYNX

The pharynx is the expanded portion of the alimentary tract lying between the oral cavity and the esophagus. It is also part of the respiratory tract, as it connects the nasal cavity with the larynx. The upper part of the pharynx, called the *epipharynx*, communicates with the nasal cavity through the choanae and is known as the *nasopharynx*.

The openings of the Eustachian tubes lying on a level with the posterior ends of the inferior nasal turbinates are to be found on the lateral walls of the nasopharynx. An accumulation of lymphadenoid tissue in the top part of the posterior wall of the nasopharynx forms the pharyngeal or third tonsil, consisting of 5-6 lobes, and diverging from a common centre. In children aged two or three years this tonsil is often hypertrophied, as they grow older it decreases in size and by the age of puberty consists of diffuse lymphadenoid tissue scarcely emerging over the surface of the mucous membrane covering the nasopharyngeal roof. A plane which is a backward extension of the hard palate separates the nasopharynx from the middle part of the pharynx, known as the *mesopharynx* or, more commonly, as the *oropharynx* (Fig. 67).

The oropharynx is bounded by the posterior and lateral walls continuous with the corresponding walls of the nasopharynx, and anteriorly it communicates with the oral

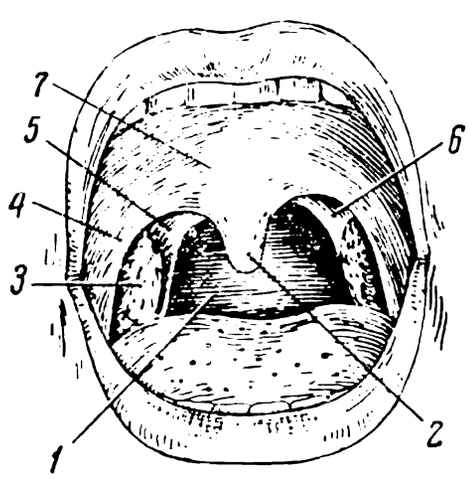


Fig. 67. Oropharynx

(1) posterior wall of pharynx; (2) uvula; (3) palatine tonsil; (4) anterior faucial pillar; (5), (6) posterior faucial pillar; (7) soft palate

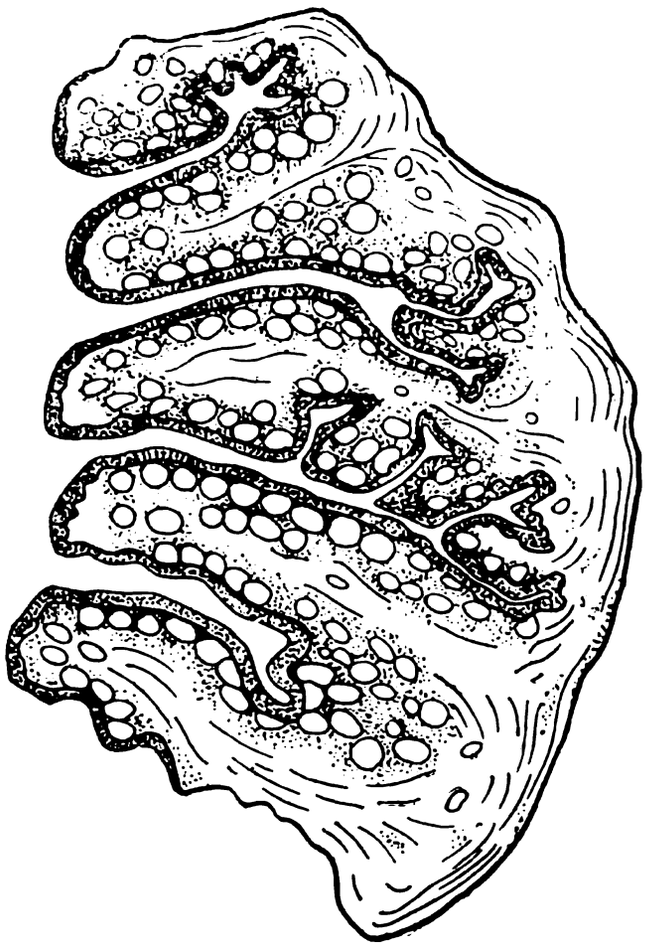


Fig. 68. Horizontal Midline Section of Tonsil Showing Deep, Branched Crypts Surrounded by Isolated Follicles

cavity through the fauces. The *fauces* are bounded by the soft palate above, by the base of the tongue below and by the anterior and posterior palatine arches, otherwise called faucial pillars, on the sides.

The pharynx contains well-developed lymphadenoid tissue which forms fairly large masses embedded on both sides between the faucial pillars, which are known as the first and second *palatine* or *faucial tonsils.* The latter's free surface facing the pharynx contains numerous pit-like depressions, or crypts, running through the entire body of the tonsil (Fig. 68). The squamous epithelium which lines the free surface of the tonsil also lines the crypts. A similar accumulation of lymphadenoid tissue at the base of the tongue forms the *lingual*, or *fourth, tonsil.* The ring-like chain of lymphoid tissue made up of these four tonsils and the lymphatic follicles in the mucous membrane is known as the lymphoid *ring* of the pharynx.

The plane that extends backwards from the base of the tongue divides the oropharynx from the *laryngopharynx*, or *hypopharynx*, which lies below and directly opens into the esophagus. The lower portion of the pharynx opens into the larynx. The mucous membrane of the nasopharynx consists of a stratified, columnar, ciliated epithelium, while its other two departments are lined with a stratified squamous epithelium. The pharyngeal mucosa contains numerous mucous glands.

The constrictor muscles lie under the pharyngeal mucosa and serve to contract the middle and lower portions of the pharynx and push food into the esophagus.

PHYSIOLOGY OF THE PHARYNX

The pharynx serves as a passage for air and food, and also as a resonating chamber for the voice. Because the digestive and respiratory tracts cross each other at the pharynx it has reflex mechanisms to regulate the passage of food and air. At rest, the nasopharynx is open to the oral cavity, whereas in swallowing and in the articulation of certain vowels and consonants the soft palate is pulled up against the posterior pharyngeal wall and firmly shuts off the nasopharynx from the oropharynx, so preventing food from getting into the nasopharynx and nose, which sometimes happens in paralysis of the soft palate, for example, following diphtheria.

The passage of the alimentary bolus is accompanied by closure of the larynx, which moves up slightly under the base of the tongue, whose pressure compresses the epiglottis against the narrow entrance to the larynx, whereupon the bolus is passed into the esophagus.

The pharynx which is studded with gustatory nerve endings on the soft palate and at the base of the tongue, also functions as the organ of taste and performs the protective function of reflex muscular contraction in response to sharp thermic and chemical irritation or to the entry of foreign bodies.

As to the exact function of the pharyngeal lymphoid ring, this is still a matter for discussion. Most authors adhere to the "protective theory", that is, regard the tonsils and the other lymphadenoid structures as a protective barrier against bacterial infection. In pathological conditions, the tonsils with their crypts which always harbour microbes prove, on the contrary, to be an entryway for infection and so account for the causal relationship between inflammations of the tonsils and constitutional diseases. In essence, the function of the lymphadenoid structures of the pharynx cannot be separated from the function of similar follicular structures in other organs, like Peyer's patches of the small

intestine, which produce lymphocytes and presumably neutralise infectious toxins entering the blood. For this reason, the removal of pathologically altered tonsils, corroborated by everyday practice, should not lead to any substantial damage from the viewpoint of their functional value for the body as a whole.

The other parts of the lymphoid ring and the lymphadenoid structures of the digestive tract will compensate for the defective function of pathological tonsils as well as for their complete removal.

The functional examination of the oral cavity and nasopharynx, the process of chewing and deglutition excluded, essentially consists in a gustatory test by using sugar, quinine, common salt and vinegar solutions to determine the taste for sweet, bitter, salty and sour substances respectively. The solution is applied on a glass stick to either side of the tongue in turn, with the nose being firmly closed to shut off the sense of smell.

In view of their different nerve-supply systems, the anterior and posterior parts of the tongue should be examined separately. The residue of the earlier used solution must be washed out of the mouth before any further examination is made.

METHODS OF EXAMINING THE PHARYNX

The oral cavity and oropharynx may be examined by means of artificial and natural lighting.

The patient and the source of light are placed in the same position as for examination of the nose; the head mirror is used similarly. Examination of the oral cavity which is a necessary prelude to examination of the pharynx commences with inspection of the lips and vestibule of the mouth. A spatula, or tongue depressor, is used to retract, in turn, the corners of the mouth and to evert with a gentle tug the upper and lower lips in order to note the colour of the mucosa, detect scratches, ulcers or fistulas and to inspect the gums and teeth. The tongue, as well as the hard and soft palates, should also be inspected. The floor of the oral cavity is examined by using the spatula to push up the tip of the tongue. The parts which are examined next are the faucial tonsils and the posterior pharyngeal wall. Here,

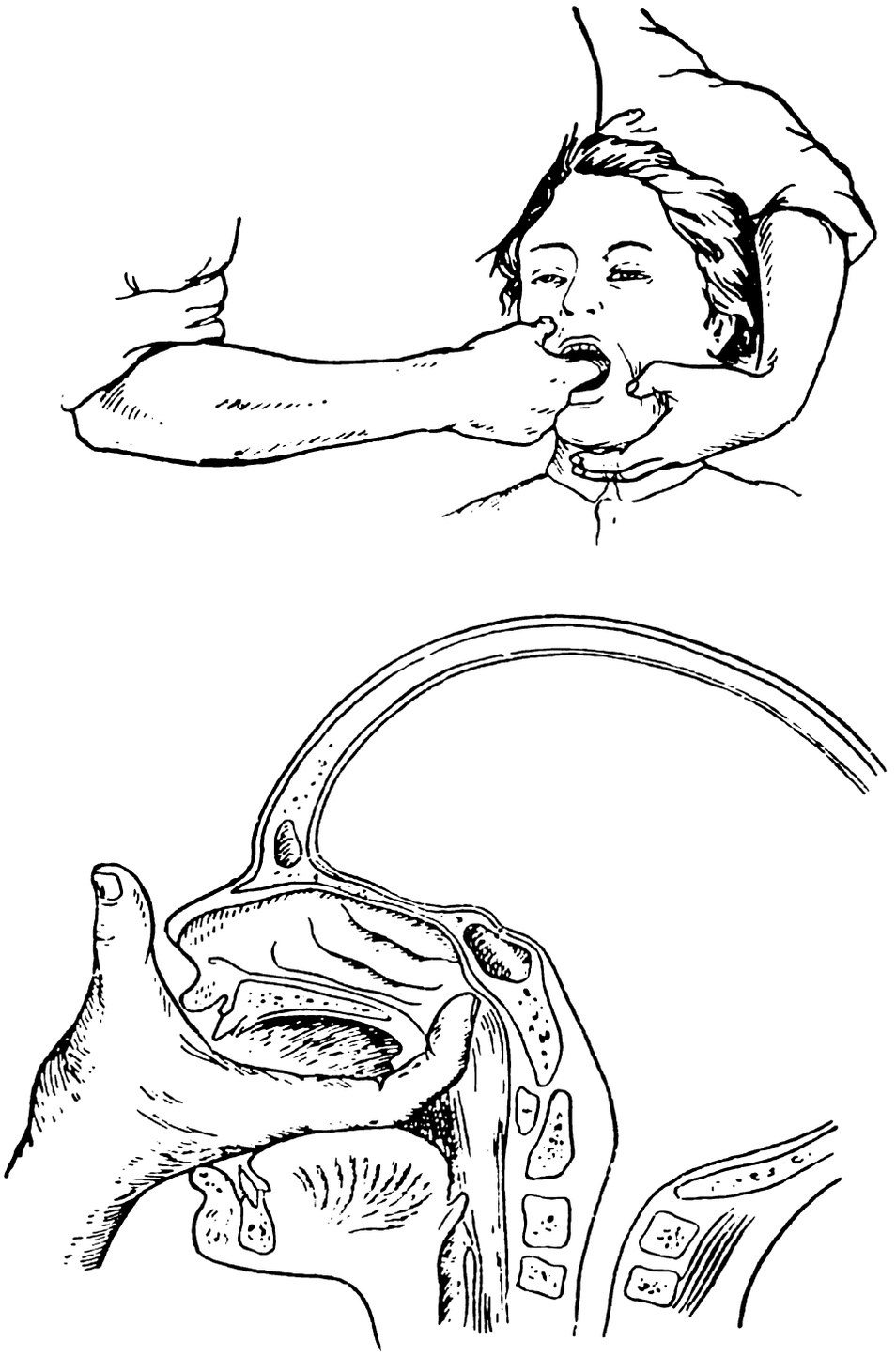


Fig. 69. Finger Palpation of Nasopharynx

the spatula is used gently to push down the dorsum of the tongue. The spatula must not be inserted too far into the mouth, or else the gag reflex will be brought into play. The patient should not put out his tongue or hold his breath, as this will interfere with the examination.

A stubborn child who resists examination by clenching its teeth must be firmly fixed in the same position as in

adenoidectomy (see Fig. 71). If it does not open its mouth for breathing when its nose is pinched, the spatula is inserted in the mouth corner, behind the posterior molar, and pushed as far as the base of the tongue. This will cause the child to gag and open its mouth, which enables the doctor to depress its tongue with the spatula in order to examine the pharynx. Attention is paid to the soft palate with its arches, the mobility of the soft palate as well as to possible fistulas, scars or fissures in the roof of the mouth. The normal colour of the pharyngeal mucosa is pink-red or pink, whereas the uvula and the faucial pillars often have a deeper hue.

In examination of the faucial tonsils, attention is given to the colour of their mucous membranes as well as to their size, possible adhesions to the faucial pillars, and the contents of the tonsillar crypts.

For inspection of the free surface of the tonsils hidden between the faucial pillars, as well as of the contents of the crypts, the anterior pillar is pulled outwards with a spatula or a blunt hook, and the spatula is gently pressed on the anterior pillar to shift the tonsil from its bed and to express the contents of its deep depressions. When the laryngeal part of the pharynx is being inspected the tongue is firmly pressed down and outwards, preferably with a curved spatula or a postnasal mirror, as in laryngoscopy.

In examination of the oral cavity and pharynx, it is essential to note simultaneously the condition of the submaxillary region and the lateral parts of the neck to detect enlarged lymph nodes, metastases or tumour outgrowths, phlegmons, etc.

Adenoid Hyperplasia (*Adenoides*)

The upper portion of the nasopharyngeal wall contains the third, or pharyngeal, tonsil which is made up of pale or red masses separated by vertical clefts. As a result of repeated inflammations in the nose and pharynx, this tonsil often hypertrophies, obstructing the openings of the choanae and Eustachian tubes and filling up a large part of the nasopharynx.

Children's infectious diseases, such as measles, scarlet fever, influenza and diphtheria, sometimes stimulate the

rapid growth of adenoids. This hyperplasia is most frequently met with in children at the age of three to eight or ten years, but it may also occur in the first year of life and after puberty, when adenoids normally tend to disappear.

Symptoms. The symptoms produced by adenoids are primarily those of nasal obstruction and continuous rhinitis which does not respond to ordinary conservative treatment. In consequence of nasal obstruction, children with adenoids sleep with their mouths open, and most of them keep it half-open when awake, which flattens their nasolabial fold and makes their facial expression apathetic and dull. Constant mouth breathing causes the hard palate to become high-arched and narrow, i.e., there forms the so-called "Gothic" palate. Hyperplasia of the pharyngeal tonsil is often accompanied by a similar enlargement of other lymphatic structures, above all, the faucial tonsils, in which case nasal obstruction becomes even more severe. This type of mouth breathing adversely affects the child's constitution, particularly the shape of its chest. That is why children with adenoids look physically weak as well as mentally retarded. They have various nervous disorders, primarily nocturnal enuresis, which ought to be regarded as a reflex neurosis. They often complain of headache and inability to concentrate.

The folds and depressions of adenoids may harbour a numerous collection of bacteria, which are responsible for frequently recurring and acute inflammations of the nasopharynx. Adenoid hyperplasia which interferes with middle ear ventilation, may cause a gradual loss of hearing or repeated inflammations in the middle ear. Owing to nasal obstruction on the side of the choanae, the child speaks with a nasal twang (*rhinolalia clausa*).

Diagnosis. Apart from the symptoms described above, the diagnosis is based on rhinoscopy, primarily postnasal mirror examination, which will offer a direct view of the nasopharynx filled with adenoid hyperplasias hanging from the roof and obstructing the choanae to a greater or lesser extent. Posterior rhinoscopy is often impossible in children, in which case the finger must be used to palpate the nasopharynx (Fig. 69). This is an easy method of detect-

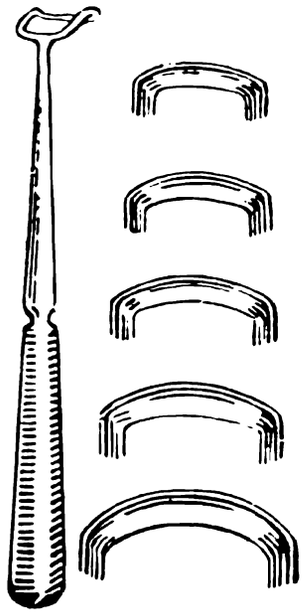


Fig. 70. Adenotomes in Five Sizes

Fig. 71. Position of Patient in Adenoidectomy

ing adenoids, as well as of determining whether they are of pasty or less soft consistency.

Treatment. In the majority of cases, adenoids are treated surgically. Indications for adenoidectomy should be sought for not so much in the size of the adenoids, as in the constitutional disturbances they are likely to cause, such as constant and stubborn rhinitis, which resists conservative treatment, and repeated inflammations of the middle ear.

The operation is usually performed in the morning when the stomach is empty by means of different types of adenotomes available in five sizes to fit the size of the nasopharynx (Fig. 70).

The child patient is wrapped in a sheet and firmly held by the assistant, who jams its legs between his own and keeps its head in position with one hand and its chest and arms with the other (Fig. 71). At operation the child should not be allowed to fidget or tilt back its head. After the tongue has been pushed down with a spatula, the adenotome is slipped into the nasopharynx behind the soft palate, set strictly along the mid-line and pressed to the nasopharyngeal roof, somewhat to the front. The adenoids are then cut off with a quick jerk from front to rear along the roof and posterior wall of the nasopharynx, and withdrawn (Fig. 72). Tags of exuberant tissue overhanging the nasopharynx are clipped off with a conchotome (Fig. 73). Bleeding is usually scanty and easily controllable.

Contraindications and *postoperative*

care in adenoidectomy. Adenoidectomy is contraindicated in hemopoietic diseases, in particular in hemophilia, and requires a preliminary blood test for coagulation.

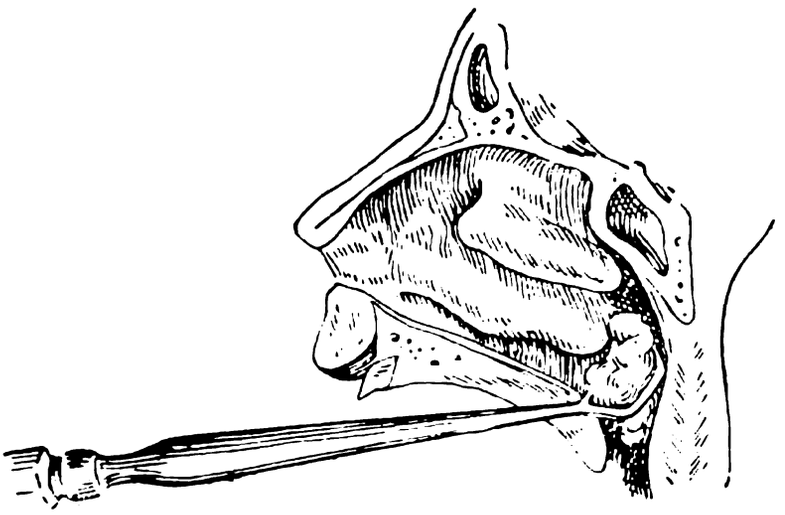


Fig. 72. Removal of Adenoids

After the operation the child is directed to clear the nose of blood by blowing it gently through one nostril at a time and to gargle the mouth with hydrogen peroxide solution. Next, the child has its nose packed with cotton wool until bleeding has ceased, is put in bed and encouraged to lie quietly for two or three hours.

If hemorrhage ensues the blood should not be swallowed but spat out into a basin.

The aftercare may be taken over by the parents who must be instructed to keep the child in bed for two or three days, give it cool and liquid meals and clean its room with a wet cloth. These measures are aimed at prevention of hemorrhage and infection of the open wound in the child's nasopharynx.

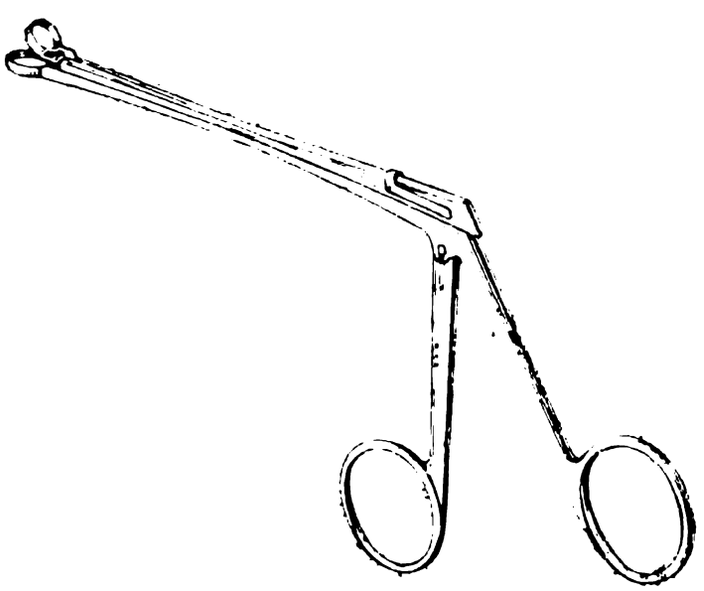


Fig. 73. Conchotome

With normal body temperature and good general condition the child may go to school on the sixth or seventh day after the operation.

The operation is soon followed by the complete restoration of nasal breathing and hearing and later by improvements in the child's physical and mental development.

Despite the removal of adenoids measures for promoting a sound constitution in children should not be neglected.

Following adenoidectomy, some children are given breathing exercises to promote nasal respiration.

If there are serious contraindications for surgical removal of adenoids, X-ray therapy may be of assistance.

Hypertrophy of Faucial Tonsils

Hypertrophy of the faucial tonsils similar to that of the pharyngeal tonsil is more common in children. In this condition, the tonsils are markedly enlarged and protrude beyond the faucial pillars (Fig. 74) or are in contact in the mid-line. Hypertrophied tonsils in children usually have no inflammatory signs. In time, these "physiologically" hypertrophied tonsils shrink to their normal size. This involution may be delayed, in which case comparatively large tonsils will be found in an adult patient.

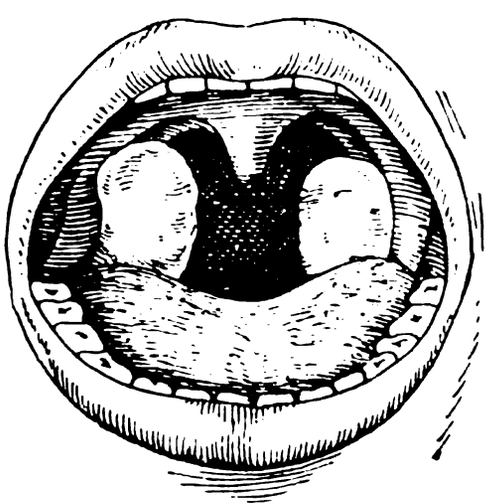


Fig. 74. Hypertrophy of Faucial Tonsils

Treatment. Health promotion is the primary measure which includes a hygienic regimen, adequate nourishment, rest in child health centres, etc. In children suffering from marked hypertrophy of the faucial tonsils which interfere with speech, breathing and the passage of food, tonsillectomy must be resorted to, the redundant tissue protruding into the nasopharyngeal space being removed with a tonsillotome (Figs. 75 and 76).

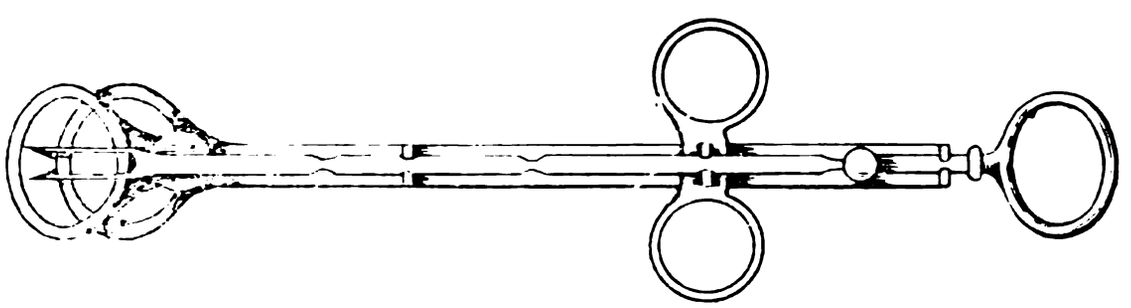


Fig. 75. Tonsillotome

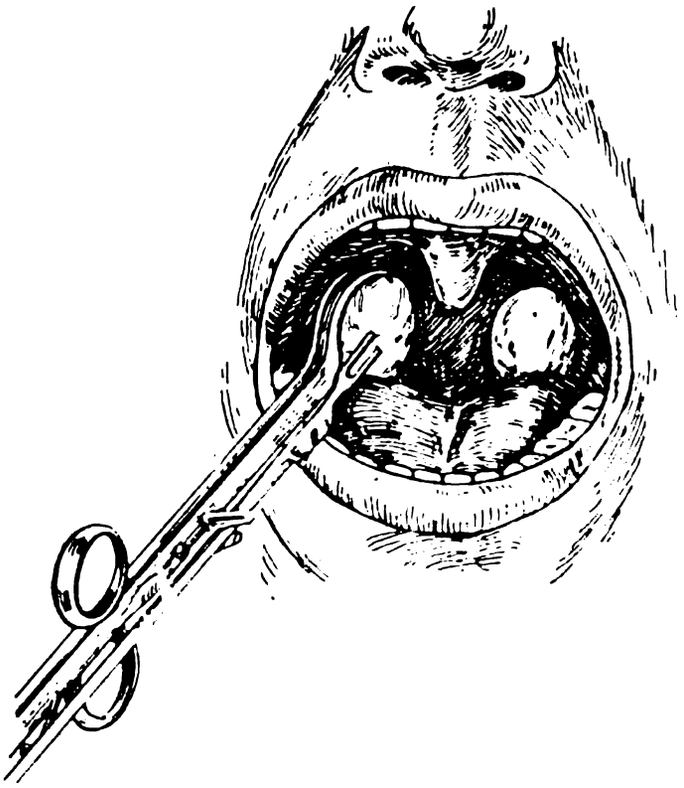


Fig. 76. Tonsillotomy

Foreign Bodies in the Pharynx

Foreign bodies enter the pharynx not only through the mouth, which is the most common route, but also through the nose as well as the larynx and esophagus. Foreign bodies that lodge in the pharynx, like fish bones, fruit stones, grains, and the like, usually enter during a meal. Among other foreign bodies found in the pharynx are fragments of dentures, coins, small toys and other objects children put in the mouth at play, as well as nails, drawing-pins, pins and buttons held in the teeth by adults at work, particularly shoe-makers and tailors.

The danger of foreign bodies slipping in with food through the mouth is greater in cases of toothless jaws with dentures which shut off the feel of the hard palate. In addition, foreign bodies often lodge in the pharynx at meals owing to a sudden cough, laughter, sneezing and talking which distract attention from chewing. Foreign bodies which become wedged in the pharynx are usually sharp and catch in the faucial mucosa, tonsils, faucial pillars or lodge at the entrance to the esophagus or larynx. Large and smooth foreign

bodies more frequently pass down to the esophageal entrance or become stuck in the esophagus itself.

Foreign bodies are less likely to lodge in the nasopharynx. They may be broken-off pieces of instruments entering through the nose, remnants of food and sometimes pinworms belched out of the stomach. Sometimes live creatures, such as leeches enter the nasopharynx through drinking filthy water from swamps and irrigation ditches.

Foreign bodies may produce different symptoms depending on their individual features. In the case of a sharp foreign body wedged in the mucous membrane, the patient will complain of a more or less severe pricking pain, particularly marked on swallowing. In other cases, a foreign body may produce mucosal inflammation with signs of hyperemia, swelling and increased secretion, which in turn cause coughing, choking and sometimes vomiting. Should a large foreign body become lodged in the lower pharynx, at the laryngeal entrance, this may produce sudden symptoms of asphyxia.

Treatment. Following location of the foreign body by a thorough and consecutive examination of all parts of the pharynx, it is extracted by aid of a straight or angular forceps with the blades in close contact. The patient is sometimes distressed by scratches and sores left by the removed foreign body which simulate the latter. Aftercare consists of mildly antiseptic gargles and a bland diet.

ACUTE INFLAMMATIONS OF THE PHARYNX

Inflammation of the pharyngeal mucosa may be acute or chronic.

Acute pharyngitis (*pharyngitis acuta*). Acute inflammations of the pharyngeal mucosa rarely occur of themselves, and are commonly provoked by a downward extension of acute catarrh of the nose and nasopharynx. Acute pharyngitis may produce different subjective symptoms in the throat, such as dryness, slight pain or tension, sore throat, etc. Physical examination reveals hyperemia of the mucous membrane, which in places is covered with mucopurulent secretion. Individual follicles exhibit as red granules on the retropharyngeal mucosa. The uvula is red, swollen and slightly edematous. Body temperature is either normal or slightly

elevated, and bodily discomfort is mild. Acute pharyngitis frequently foreshadows or accompanies tonsillitis.

Treatment. This is similar to that in tonsillitis, namely, rest in bed, a bland diet of warm and liquid or semi-liquid food, administration of salicylic and sulfonamide drugs, application of a hot compress to the neck and use of the following antiseptic gargles:

Rp. Dec. radicis Althaeae 400.0
Natrii benzoici 6.0
Glycerini 10.0
T-rae Opii benzoici 3.0
MDS. Gargle

Rp. Kalii hypermanganici 1.0
DS. Two or three crystals in a glass of water for a gargle

The gargle should be warmed a little beforehand and the head tilted back to let the liquid irrigate the farthest portions of the pharynx.

Catarrhal, Lacunar and Follicular Tonsillitis

Tonsillitis is a generalised infectious disease with inflammation of the pharyngeal lymphoid structures, above all the faucial tonsils, and with swelling of the regional lymphatic glands.

The inflammation in the pharynx presents a wide variety of morbid symptoms owing to the presence in the pharyngeal mucosa of adenoid tissue which may be wholly or partly involved in the process. The inflammation may be diffuse and superficial when it affects the surface of the tonsils and their crypts, or it primarily involves adenoid tissue, tonsillar parenchyma, with the follicles embedded in it.

The cause of tonsillitis is invasion by streptococci and, less frequently, by staphylococci and pneumococci, while the predisposing factors are thermic, chemical and mechanical irritations of the mucosal receptors of the fauces and tonsils in the presence of a diminished body resistance. The infection may be introduced by external factors or be activated by an increased virulence of the germs permanently inhabiting the tonsillar crypts, mouth and pharynx.

The source of infection may be a suppuration in the nose and paranasal sinuses or dental caries.

Tonsillitis often develops as a grave constitutional disease, which is evidenced by frequent complications in such organs, as the heart, joints and kidneys, as well as by the fact that the local signs in the fauces are often preceded by severe malaise with fever, headache and rheumatic pain.

Tonsillitis may occur in the catarrhal, lacunar and follicular forms depending on its severity and on the presence or absence, as well as the character and location, of a patchy membrane on the faucial tonsils.

Catarrhal tonsillitis. The patient first complains of dryness and soreness in the throat and later of moderate throat pain on swallowing. The temperature is usually subfebrile, whereas in children a high-grade fever is more common, and vomiting is one of the early symptoms. Headache and feebleness are frequent. The disease continues three to four days. In mild cases, examination of the pharynx reveals slightly swollen and red tonsils whose surface is coated with a yellowish mucoid secretion. In severe cases, there is punctate hemorrhage on the mucosa as well as edema of the uvula and faucial pillars. The lymph nodes below the mandibular angle are swollen and tender.

Catarrhal tonsillitis is nearly always present with many acute infectious diseases. In scarlet fever, it takes a peculiar course. At the onset, the inflammatory redness is sharply outlined in the centre of the soft palate, whereas in unaccompanied tonsillitis the tonsillar mucosa is most affected. Within a day or two, the bright and limited redness caused by catarrhal tonsillitis present in scarlet fever disappears, and hyperemia extends over a wider area invading the tonsils and the posterior wall of the pharynx.

The characteristic skin rash of scarlet fever appears after the first 23 or 24 hours. In measles, hyperemia of the fauces is of spotted character and appears on the second or third day after the onset of the disease as part of general enanthema.

In *lacunar tonsillitis*, the general symptoms are more pronounced. The constitutional disturbance is more severe than in catarrhal tonsillitis, while pain felt on swallowing and headache are worse. There is a marked fever which in

children rises as high as 40 °C. The disease often disappears on the fourth or fifth day. The pain felt in the throat shortly after the onset of the initial symptoms becomes especially severe on swallowing and is sometimes referred to the ear. Debris consisting of bacteria, desquamated epithelial cells and leukocytes fills the crypts and spreads in white and yellow patches on the slightly swollen and red tonsillar mucosa. A continuous coat made up of yellowish-white membranes or patches often covers the free surface of the tonsils without extending beyond their limits. In such cases, there is marked congestion and considerable edema in the soft palate and faucial pillars. In lacunar tonsillitis affection of the tonsillar parenchyma causes the swelling and hypertrophy of the tonsils. The formation of patches in the lacunar mouths distinguishes this disease from diphtheria, where the initial lesion is confined to the eminent parts of the tonsillar mucosa. The regional lymphatic glands are swollen and tender. Lacunar tonsillitis is a highly-contagious acute infectious disease, which is proved by its frequent outbreaks in families and hospitals.

Follicular tonsillitis (coloured Table III, Fig. 1). This condition is another form of the same infectious disease occurring independently or concomitant with lacunar tonsillitis. The red and swollen mucosa of both tonsils becomes the site of eruption of a considerable number of round and slightly prominent yellow or yellowish-white nodules the size of a pin-head, which are suppurated tonsillar follicles. This disease differs from other forms of tonsillitis with patches in that the suppurated follicles are of uniform size and regular shape, and are confined to the free area of the tonsils. In the process of suppuration, the yellowish-white follicles gradually grow in size and burst into the pharyngeal space. Evacuation of their contents into the connective tissue bed of the tonsil may produce a peritonsillar abscess.

The onset of follicular tonsillitis is often sudden, accompanied by a chill, a rapid rise in temperature to 40 °C or higher, a sore throat, pain in the back and limbs, headache and a general malaise. The submandibular lymph nodes are swollen and tender where the patches are heavier, as in lacunar tonsillitis. This condition is marked by a sharp

increase in the number of leukocytes, mainly neutrophils, with a leftward shift in the white cell count.

Follicular tonsillitis may attack the pharyngeal and lingual tonsils simultaneously. Though its course is severe this disease usually ends in complete recovery in five to seven days' time.

Tonsillitis is liable to produce a number of complications of a local and general character.

The former refer to peritonsillar abscesses and acute otitis media, the latter—to cardiac and articular *rheumatism*, *renal diseases* and *sepsis*.

Diagnosis. This disease is not difficult to recognise. In diagnosing lacunar tonsillitis particular care should be taken not to mistake it for diphtheria. The difference between them will be discussed in the section on diphtheria.

In tonsillar syphilis, there is very little malaise, moderate pain on swallowing, and a very slight fever. The faucial pillars and tonsils are bright red and covered with greyish-white round papules which are also found on the mucosa of the cheeks, gums and tongue. Numerous swollen and solid lymph nodes can be felt under the mandible and on the neck. The diagnosis of tonsillar syphilis is verified by general examination and Wassermann's test.

Prognosis is usually favourable, but it is rather common that recovery from tonsillitis, however severe, is followed by grave constitutional complications, like rheumatoid arthritis, rheumatic endocarditis, nephritis or septicopyemia. The forecast, therefore, should be made with caution, particularly in cases of recurrent tonsillitis followed by complications.

In all cases of more or less severe tonsillitis, blood tests should be performed since this condition may be a symptom of a serious disease of the blood, leukemia, etc.

Treatment. The patient is advised to take to bed, as a prophylactic measure against complications, is prescribed a diet of thin gruel and given acetylsalicylic acid (aspirin) with caffeine orally.

Rp. Aspirini 0.5
Coffeini natrio-benzoici 0.1
M. f. pulv.
D. t. d. N. 10
S. One powder dose two or three times daily

Small children are given a solution of sodium salicylate. Mildly antiseptic gargles, like boric acid, sodium benzoate and other solutions, are used for throat irrigation.

Rp. Hydrogenii hyperoxydati 3% medic. 100.0
DS. One or two tablespoonfuls in a glass of water for a gargle

Rp. Acidi borici 25.0
DS. One teaspoonful in a glass of warm boiled water for a gargle

Rp. Gramicidini 2.0
D. t. d. N. 6 in amp.
S. The contents of one ampule dissolved in a glass of water for a gargle

Rp. Natrii benzoici 25.0
DS. Half a teaspoonful in a glass of water for a gargle

Children are given plenty of warm drinks. A hot compress on the neck, preferably soaked in a solution of one-third of alcohol to two-thirds of water, is effective as well as steam inhalations from a hot 2% baking soda solution and administration of laxatives. The condition of the heart and kidneys should be watched, in the case of the latter—by analysis of urine.

The hot compress will help diminish infiltration and congestion in the pathologically changed organ, viz., in the tonsils, after which the morbid process will speedily resolve, and pain will be alleviated.

The streptococcic nature of tonsillar infection is an indication for the use of streptocide and other sulfonamide drugs as well as penicillin by intramuscular injection, and biomycin and terramycin in tablet form. Streptocide, sulfathiazole and sulfadimezin are given for a few days in 0.3 to 0.6 g doses five to six times a day. Children are given accordingly smaller doses.

Prophylaxis. The prevention of tonsillitis is based on general rules for strengthening the body and enabling it to resist various harmful external influences.

These rules are as follows:

1) Observance of sanitary and technical regulations in industry, selection by fitness for a particular job, registra-

tion and regular examination and treatment of persons susceptible to tonsillitis;

2) more hygienic living conditions, personal hygiene and body hardening;

3) treatment of diseases conducive to tonsillitis, such as chronic tonsillar hypertrophy, purulent highmoritis, adenoid hyperplasia, dental caries, etc., which affect the upper respiratory tract.

Although tonsillitis infection is not highly contagious, it is advisable to isolate the patient with tonsillitis and provide him with individual eating and other utensils. Healthy persons, children in particular since they are especially susceptible to the disease, should avoid contact with the patient.

Acute inflammation is not always restricted to the tonsils, but may often involve the entire lymphadenoid tissue of the pharynx; yet lesion of individual tonsils may have peculiar clinical symptoms of its own.

Acute pharyngeal tonsillitis. This condition commonly occurs in children, particularly in cases of hypertrophy of the pharyngeal tonsil, and is called acute adenoiditis or retronasal tonsillitis (*angina retronasalis*). The symptoms are fever, a general feeling of weakness, and signs of rhinitis. The troublesome smarting sensation in the nasopharynx is accompanied by cough and otalgia. The cervical lymph nodes are often swollen and tender.

Treatment is the same as that for acute rhinitis.

Acute lingual tonsillitis. This condition is much rarer. Its general symptoms are similar to those of other types of tonsillitis: local pain on swallowing, marked tenderness at the base of the tongue on pressure with a spatula, a bright red and swollen lingual tonsil, sometimes covered with yellowy dots or coated all over as in lacunar tonsillitis.

Treatment. This is similar to that applied to inflamed faucialtonsils.

Ulcero-membranous angina (Vincent's angina). This disease causes ulceration of the mucosa of the soft palate and tonsils, less commonly of the gums, retropharyngeal wall and cheeks. It is due to the symbiosis of a fusiform bacillus and an oral spirochete which usually resides in a low virulent state within the folds of the oral mucosa.

The condition is marked by superficial necrotic patches of a yellowish-white colour on the mucosa of the tonsils and soft palate. These necrotic patches quickly merge to form superficial and sometimes deeper ulcers on the mucosa, which are not very painful but often produce bad breath and salivation. The temperature is usually subfebrile or normal with little constitutional change. The regional lymphatic glands are swollen and tender.

The normal duration of the disease is about a week, but it may linger for a few weeks and longer.

Ulcero-membranous angina occurs either sporadically or in epidemics. When observing an outbreak of this disease in Finland in 1888, S. P. Botkin, the famed Russian internist, called it "Finnish quinsy".

N. P. Simanovsky, the first Russian to have written a treatise on this subject, in 1890 described a similar epidemic of ulcero-membranous angina in St. Petersburg.

According to earlier evidence of Russian physicians, namely, Filatov and others, ulceration of the oral cavity and pharynx occurs with ulcerative stomatitis. Thus, the identity of ulcero-membranous angina with ulceration of the oral mucosa had been proved clinically long before Vincent discovered the pathogen of this disease in 1898.

Diagnosis. The diagnosis is verified by a laboratory investigation of smears taken from the ulcer contents, which reveals both fusiform bacilli and spirochete. Sometimes, external evidence is insufficient for differentiation of ulcero-membranous angina from diphtheria, syphilis or cancer.

Treatment. In mild cases, the disease will have run its course in a few days' time after treatment with mild antiseptic gargles of dilute hydrogen peroxide, potassium chlorate, boric acid or potassium permanganate. In more severe cases with extensive ulceration and a high-grade fever, a solution of salvarsan may be used to paint the ulcers, or may be given intramuscularly. The most efficacious remedy, however, is intramuscular injection of penicillin.

Agranulocytic angina (*angina agranulocytotica*). This condition is usually marked by a sudden fever, sometimes accompanied by chills and sore throat. Examination of the throat will reveal a markedly swollen mucosa covered with necrotic patches of a dirty grey colour. Deep ulcers form

later which are not confined to the tonsils but spread all over the pharynx, oral cavity and larynx. Constitutional disturbance is grave, and fever is of the septic type.

The blood picture in such cases will nearly always reveal a drastic decrease and sometimes nearly complete absence of granular leukocytes, or granulocytes, from the blood. The number of white cells in the blood drops from between 6,000 and 7,000 to 500 per cu mm and less. In the absence of blood-test evidence this disease may be mistaken for diphtheria and ulcero-membranous angina.

This exceedingly grave disease is comparatively rare, and the precipitating causes are still unknown. It lasts four to five days or up to several weeks and if untreated frequently proves fatal.

Treatment. This is by blood transfusion, X-ray irradiation of the bone marrow, drug therapy to speed up leukocytosis and with pentoxyl given in 0.3 g doses four times daily. Local treatment includes gargling as well as painting the ulcers with a 5% cocaine solution and dusting them with orthoform or anesthesin powders. The wide use of intramuscular penicillin injections has given medicine a powerful weapon to combat this disease whose outlook formerly was nearly hopeless.

Tonsillitis in Infectious Mononucleosis and Listeriosis

(*Angina monocitotica et listerellosa*)

Infectious mononucleosis is basically a sporadic disease, though, less frequently, it may occur in epidemics confined to a small community or family. The clinical picture of this condition was first described by N. F. Filatov in 1885 as glandular fever characterised by swollen lymph nodes and distinctive changes in the blood.

Etiology. Recent clinical and bacteriological studies suggest that this disease is caused by a virus. Some authorities believe that infectious mononucleosis is a form of listeriosis caused by bacteria of the genus Listeria (*Listerella monocitogenes*). This conjecture seems reasonable in view of the similarity that exists between the clinical symptoms of infectious mononucleosis and listeriosis. In the former

case, the disease runs a milder course with fewer of the grave complications fairly common to listeriosis.

The clinical picture and course. Infectious mononucleosis occurs mainly in young people between 10 and 30 years of age and is caused by droplet infection. The disease has various, sometimes light, clinical forms. In typical, more frequent, cases, the onset is marked by rigor, headache and a high fever of 39° to 40°C, which later becomes remittent. The most characteristic symptom is one of irregularly swollen and tender cervical, submandibular, and other lymph nodes with a simultaneous enlargement of the spleen and sometimes of the liver.

Tonsillitis in infectious mononucleosis usually appears on the third or fourth day in a broad variety of forms. It may be catarrhal, lacunar, and quasi-membranous resembling diphtheria, as the patches spread over the faucial pillars, uvula and soft palate. More severe cases may develop into the ulcero-necrotic form with deep ulceration in the body of the tonsil coated with a greyish-green membrane and foul odour from the mouth. These changes in the pharynx cause excruciating pain and considerable enlargement of lymph nodes.

Diagnosis. This is based on an examination of the blood which makes it possible to differentiate this condition from leukemia, agranulocytosis, tularemia and brucellosis. The blood picture shows a moderate leukocytosis (15,000-20,000) with a predominance of mononuclear cells, which may number 50 to 90 per cent of the total leukocytes. The red cell count is usually normal.

The disease lasts two or three weeks, then the fever gradually subsides, local symptoms of tonsillitis disappear, pain ceases, the spleen shrinks to its normal size and the swollen lymph nodes gradually diminish to become impalpable. However, the blood condition rises to normal more slowly.

Treatment. This is symptomatic. Antibiotics, such as penicillin, as well as hypodermic injections of arsenic and intravenous injections of ascorbic acid with glucose are recommended to ward off secondary infection. The use of sulfonamide drugs which adversely affect hemopoiesis should be avoided.

Septic angina (alimentary toxic aleukia). The onset of this disease is marked by a sudden fever of 39° to 40°C, inflammatory and necrotic signs in the throat, petechial eruptions and severe hemorrhage from the nose and mouth.

The anginal stage is not the onset of the disease and follows food intoxication that has been in progress for one to three weeks without any significant signs.

The disease is caused by cereal food such as millet, wheat, rye, barley, buckwheat, and oats, that had been left out in the field during the winter.

Ingestion of this grain, in particular millet, will cause a bitter taste and a burning sensation in the mouth, pharynx, esophagus and stomach, as well as numbness in the tongue. These symptoms are often accompanied by nausea, vomiting, and headache. Further consumption of this food, however, does not cause a recurrence of these sensations since the body seems to become immune.

Yet in other cases, the absorption of this food for only two or three weeks is followed by headache, prostration and weakness. Punctate hemmorrhage looking like flea bites appears on the skin. Already at this early period of septic angina, blood analysis will reveal a progressive reduction in the leukocyte count, viz., onset of the period of leukopenia.

Should consumption of this winter-spoiled grain not be discontinued, and treatment not be started immediately, then within one, two or three weeks there will be a sudden onset of septic anginal syndrome. The whitish or yellowish-brown membrane which appears on the tonsils marks the onset of necrosis which soon, in fact in 24 hours, causes deep ulcers that bleed readily. This ulceration commonly affects not only the tonsils which soon collapse completely but other aggregations of lymphadenoid tissue as well, and may extend to the palatine, pharyngeal and esophageal mucosa and, sometimes, to that of the oral cavity.

Withdrawal of toxic products from food at the initial period of the disease, prior to the onset of anginal symptoms, may often bring recovery, especially if the total amount of toxic food eaten has been moderate.

Advanced septic angina is frequently fatal.

Medical aid for this condition essentially consists in an

early diagnosis of septic angina in the leukopenic stage, a mass examination of inhabitants of districts where winter-spoiled grain was used for food and an indispensable analysis of blood to detect leukopenia.

Treatment. At the first signs of the disease, toxic products should be immediately withdrawn from food, and lavage of the stomach undertaken. The patient is then given large doses of magnesium sulfate or sodium sulfate to cleanse the stomach of toxic food residue. The diet must be nourishing and rich in proteins and vitamins, and drink must be given in plenty to help expel toxins from the body. Local treatment, apart from the use of gargles and anesthetic ointments, is by sprinkling the ulcerated surfaces with streptocide or sulfadimezin powders twice daily.

Intramuscular penicillin injections and pentoxyl have been used with success. Hemorrhage can be checked by blood transfusion in 200-300 ml doses and by intravenous injections of calcium chloride. Cardiac failure may be helped by administration of camphor, caffeine and strychnine.

Prophylaxis. The basic means of control of septic angina is prophylaxis by timely and careful harvesting of all cereals and by making the public understand how dangerous it is to use winter-spoiled cereals for food. The chemical nature of the poison responsible has not yet been established, and only its heat resistance is obvious, since neither cooking, nor boiling or any other culinary treatment of winter-spoiled grain can reduce its toxicity.

Peritonsillar Abscess or Quinsy

(*Angina phlegmonosa, s. peritonsillitis abscendens*)

The peritonsillar abscess is a fairly common sequel to chronic tonsillitis and, more frequently, to acute tonsillitis. The infection penetrates from the depth of a tonsil crypt into the connective tissue bed of the tonsil causing an inflammatory process which leads to the formation of an abscess.

Symptoms. The commonest complaint in quinsy is a spontaneously growing pain. The patient who has just recovered from tonsillitis again feels pain on swallowing, mostly unilateral and has a rising fever.

Half of the soft palate becomes markedly red and increasingly swollen, gradually hiding the tonsil and pushing the edematous uvula to the opposite side (coloured Table III, Fig. 2).The pain is more severe than in lacunar or follicular tonsillitis, and is felt even when the patient is motionless. It becomes a sharp shooting pain, frequently radiates to the ear and increases on swallowing, coughing, or any movement. The mouth is opened with pain and difficulty and the patient inclines his head towards the sore side. The regional lymphatic glands on the affected side are often swollen and tender. The almost complete immobility of the soft palate makes the voice muffled and the swallowing of food difficult or even impossible.

The abscess forms in the connective tissue bed of the tonsil, commonly above and before or behind the tonsil (Fig. 77, a). It may also develop in connective tissue at the lower pole of the tonsil and anteriorly to it.

In the event of an anterosuperior abscess the most marked redness is in the soft palate and the anterior faucial pillar. An abscess behind the tonsil (Fig. 77, b) is identified by an oval-shaped and extremely tender swelling and redness in the posterior pillar, with the soft palate and the anterior pillar showing no marked changes. The abscess comes to a head in five to seven days. Its progress is accompanied by a characteristic throbbing pain and increased infiltration

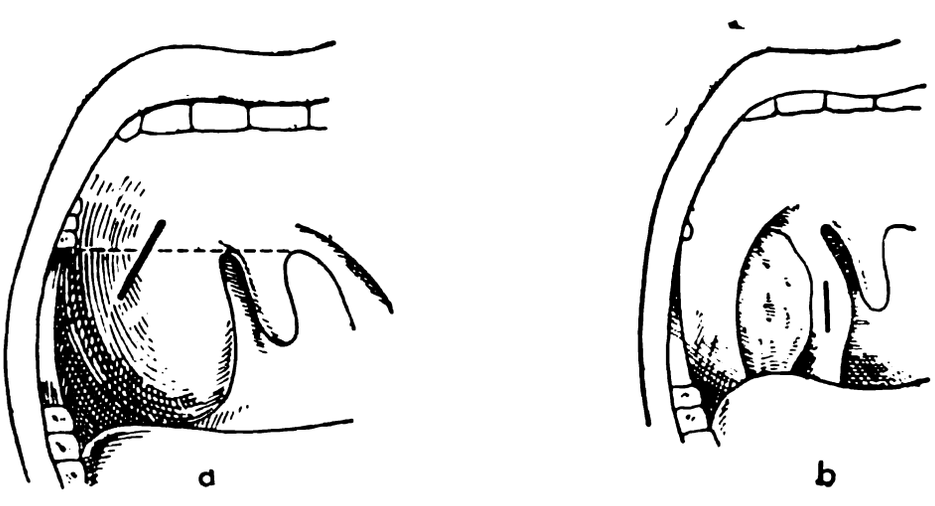


Fig. 77. Peritonsillar Abscess

(a) anterosuperior peritonsillar abscess; (b) posterior peritonsillar abscess. Line indicates site of incision

with softening in a particular place. Frequently the abscess ruptures through the anterior pillar or drains above through the tonsil.

The opening of the abscess between the anterior pillar and the edge of the tonsillar capsula is often inadequate for its full evacuation and should therefore be enlarged to assist recovery.

Diagnosis. Identification of quinsy involves no special difficulties. Peritonsillar abscess is commonly preceded by tonsillitis where unilateral redness in the fauces gradually expands, while swelling and increasing pain with a high-grade fever serve as another pointer to the genuine cause of the condition.

Treatment. This depends on the stage of inflammation. In the beginning, at the stage of infiltration, it may be possible to abort the process by adequate sulfonamide therapy in a total dose of 3 to 6 g per 24 hours. In progressive abscess formation the use of sulfonamides or penicillin in inadequate doses sometimes retards the process and prevents the ripening of the abscess, whereupon no regression of inflammation in the peritonsillar connective tissue is seen for two and even three weeks. Intramuscular injections of penicillin in comparatively large, 50,000-100,000 unit doses given six to eight times per 24 hours bring about a decided turn for the better already within the first score of hours and full recovery later. Synthomycin may be substituted for penicillin in 0.5 g doses given in capsules four times daily during four or five days until complete disappearance of the inflammatory infiltration. A decrease in inflammatory infiltration often leads to the formation of a markedly encapsulated abscess, and quick recovery ensues after this has been lanced. All kinds of heat treatment are given to speed up resorption of the infiltrate and the ripening of the abscess, particularly in cases where antibiotics are not used. Hot compresses are applied to the neck, as well as inhalations of 2% baking soda solution with an inhaler once every two or three hours, warm gargles of diluted boric acid, potassium permanganate, baking soda, etc.

The patient should be given a diet of warm liquid or gruel. Severe pain may be relieved by aspirin (with caffeine)

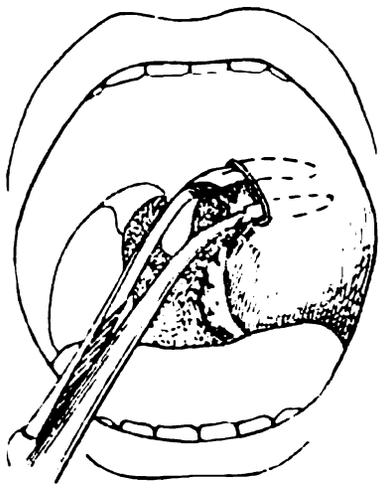


Fig. 78. Opening of Anterosuperior Peritonsillar Abscess through Supratonsillar Fossa

given in 0.5 g doses several times a day. Luminal in an 0.1 g dose, or morphine or bromine drugs are given at bedtime.

The ripe abscess may often be opened with a blunt probe or bent forceps via the supratonsillar fossa upon rupturing the tonsillar capsula (Fig. 78). Incision of an anterior peritonsillar abscess is made in the anterior faucial pillar, 1 to 2 cm outwards from its edge, and over the most protruding and softened portion. Where the softened area cannot be detected, it is recommended that the incision be carried out along the mid-line between the base of the tongue and the posterior molar to a depth of 1 or 2 cm.

Among the complications of quinsy is hemorrhage due to pus corroding the walls of the pharyngeal vessels, deep cervical phlegmons, edema of the larynx, and sepsis.

Prophylaxis. Oral hygiene is extremely important as well as the treatment of chronic tonsillitis, dental caries and suppurations in the paranasal sinuses. Frequent recurrence of peritonsillar abscesses necessitates tonsillectomy, i. e., complete excision of the tonsils three or four weeks after recovery from quinsy.

Where indicated, however, the presence of a peritonsillar abscess is no obstacle to removal of the faucial tonsils which, in particular, is a prophylactic against recurrent abscess formation.

Lingual Tonsil Abscess (*periamygdalitis abscendens lingualis*). Inflammations or injuries of the lingual tonsil may cause phlegmons at the base of the tongue similar to phlegmons in the faucial tonsils. In this condition, there is always severe malaise, high-grade fever, headache, acute pain on swallowing and speaking. At first, the base of the tongue grows red, swollen and edematous. The edema may sometimes encroach on the entrance to the larynx and epiglottis and threaten death by suffocation. The abscess may develop towards a side of the neck causing tender swelling

and redness in the floor of the oral cavity or at the mandibular angle.

Treatment. This condition is treated in the same way as the peritonsillar abscess.

Retropharyngeal abscess (*abscessus retropharyngealis*). This abscess is most commonly encountered in emaciated and weak infants and young children. It arises owing to suppuration of the lymph nodes lying on the posterior pharyngeal wall in front of the spinal column, in the so-called retropharyngeal space. The infection enters here through lymphatic channels from the nasal cavity, sometimes after the ordinary common cold, or from the oral cavity through fissures and abrasions caused by improper care of the mouth in babies. In children aged above five or six years retropharyngeal abscesses are rare because by this age the prespinal lymph nodes have usually atrophied.

Symptoms. In small children the abscess causes a high fever of up to 39-40°C. Because swallowing is painful the child will choke and refuse food which may often get into the nose and larynx. In addition, there is marked nasal obstruction, muffled voice and stertor, especially heavy in sleep. It is the latter symptom that usually alarms the parents and gives the doctor a clue to diagnosis. If a retropharyngeal abscess is suspected, examination of the posterior pharyngeal wall alone is often insufficient. Therefore, the pharynx and nasopharynx should be palpated with the finger and a pasty, fluctuating swelling commonly located somewhat higher than the soft palate and closer to one side of the pharynx will be felt. The abscess seldom lies in the horizontal mid-line of the pharynx or in its lower part. The regional lymphatic glands are usually swollen on the affected side. Adults may have abscesses of infiltrative character on the posterior pharyngeal wall, for example, in tuberculous and syphilitic spondylitis. In such cases the course is torpid and the fever is low, the neck is rigid, and pain is felt on turning the head.

Treatment. Incision of the abscess is indicated as soon as the diagnosis is made, in order to avoid spontaneous rupture with the danger of suffocation by pus suddenly filling the larynx, especially in sleep, or pneumonia due to pus aspiration into the lower respiratory tract.

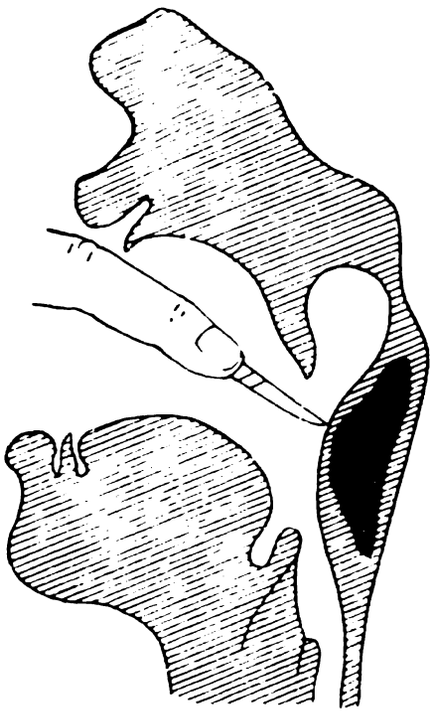


Fig. 79. Opening of Retropharyngeal Abscess

A small incision is made in the abscess with a scalpel bound with an adhesive plaster 0.5 cm from its end (Fig. 79). The opening is dilated with forceps, and the child's head quickly pushed down and forward to divert pus from the larynx. Two or three days later, it is often necessary to draw apart the closed lips of the wound and evacuate the accumulated pus. After the abscess has been opened, recovery is usually speedy.

Infiltrative abscesses must not be opened on the pharyngeal side as there is a danger of secondary infection and formation of a fistula. They are treated by punctures and suction followed by infusion of 5 to 10 ml of iodoform emulsion or streptomycin solution.

Faucial Diphtheria
(*Diphtheria faucium*)

Diphtheria is an acute contagious disease caused by Loeffler's bacillus, whose clinical signs occur between the second and seventh day of infection. The disease is communicated either by direct contact when the patient sprays sputum particles in cough, sneezing and talking, or by indirect contact through objects he has handled. Infection may also be transmitted by recovered and healthy diphtheria carriers. The disease most commonly affects the fauces and tonsils, and primarily occurs in children aged between two and six years.

In breast-fed babies and adults faucial diphtheria is a comparatively rare phenomenon.

The clinical picture and severity of the disease varies, and it is, therefore, subdivided into the localised, diffuse, toxic and hemorrhagic forms.

The mild forms, such as localised diphtheria, may be similar to lacunar or follicular tonsillitis, the only difference being in body temperature, which is subfebrile and in

adults may often be normal. This condition is characterised by the formation of a greyish or greyish-white patchy membrane on the moderately swollen and congested surface of the tonsils and a slight constitutional disturbance. Pain in the throat is not severe. The submandibular lymph nodes are only swollen slightly, and the disease may disappear within three or four days.

Usually the individual spots on the tonsils soon merge to form whitish, greyish-white or, less frequently, yellowish islands which grow into compact, adherent crusts. The latter may not only occupy the free surface of the tonsil, but, in the so-called diffuse form, may invade the faucial pillars and soft palate and often rise above the surface of the mucosa (coloured Table III, Figs. 3, 4). In the event of the membrane being stripped off the tonsil bleeding will ensue.

The severe forms, otherwise known as toxic, cause a much graver constitutional disturbance with a high fever of 39-40°C, pallor, apathy and bad breath. The weak, fast, often arrhythmic pulse is indicative of a toxic heart affection. Already in the early days of the disease the markedly swollen and reddened tonsils become coated with an extensive dirty-grey membrane which tends to grow and often spreads down to the larynx and trachea, in which case the condition is called descending croup. The lymph nodes are swollen and the cervical cellular tissue is edematous. The sites of diphtheritic process sometimes become necrotic, the membranes take on a dirty-grey, dark colour, and a fetid, sanious discharge appears from the oral and nasal cavities.

In the hemorrhagic form of diphtheria, there is hemorrhage in the mucous membranes and on the skin, while the membraneous patches become saturated with blood.

In the toxic form, where edema of cellular tissue spreads down to the clavicle and below, as well as in the hemorrhagic form, it is not always possible to save the life of a child patient. In the diffuse form without edema of the cervical cellular tissue, recovery is certain, if serum treatment was started in time.

It is essential to distinguish the localised diffuse form of diphtheria from lacunar tonsillitis, especially when the latter causes extensive membrane formation. To facili-

Table of Distinctive Symptoms of Diphtheria and Lacunar Tonsillitis

Symptoms	Tonsillitis	Diphtheria
Swollen tonsils	Less marked than in diphtheria. More frequently it is bilateral	More severe, accompanied by edema of the faucial pillars, uvula and soft palate. May be unilateral
Patches	Spread within free areas	Extend beyond tonsils to faucial pillars, soft palate and posterior pharyngeal wall*
Colour of patches	Yellowish	White, greyish-white, dirty grey
Adherence of patches	Patches superficial and peel off easily	Patches deep, with necrosis of mucosa; in typical cases strip off with difficulty to leave a bleeding surface
Pain on swallowing	Sharp	Not always marked
Regional lymph glands	Swollen, individual nodes easily palpated and extremely tender	Markedly swollen nodes on both sides from early days of disease, edema of subcutaneous tissue; flattened out contours of neck
Constitutional disturbance	Less severe than in diphtheria	Increasingly severe in toxic form
Fever	Within 39-40°C	From subfebrile to 40°C; more stable
Pulse rate	Corresponds to fever	Slow at first, then fast, superficial and arrhythmic
Results of bacteriological examination	Negative (for Loeffler's bacilli)	Positive in most cases

* Where the patches have spread beyond the free surface of the tonsil, scarlet fever must be excluded by an examination of the skin, and ulcero-membranous angina.

tate differentiation between faucial diphtheria and tonsillitis we have drawn up a table listing the characteristic symptoms of both diseases (see page 190). In this scheme each symptom taken separately is not pathognomonic of either disease, whereas judgement based on the sum total of symptoms available may help diagnosis in obscure cases.

A high fever without obvious bodily discomfort is more common in tonsillitis, while a moderate fever of over 37 to 38 °C with marked malaise will be suggestive of diphtheria.

In tonsillitis, the membranous patches are more superficial and can easily be removed, whereas in diphtheria the thicker membranes rise above the mucosal surface and are difficult to remove. Tonsillitis membranes rarely extend beyond the limits of the free tonsil surface. Where possible a throat swab should be taken to ascertain the presence of diphtheria bacilli.

Treatment. When verifying the diagnosis and in uncertain cases, an antidiphtheric serum should be given immediately without waiting for the bacteriologist's report.

The serum is usually warmed and injected intramuscularly. In the localised form of faucial diphtheria the serum dose is 5,000-8,000 a.u. (antitoxic units).

In the diffuse form, the initial dose is 10,000-15,000 a.u.; severe cases of toxic diphtheria are given a single primary dose of 30,000 a.u., while the next dose is given in 12 or 24 hours' time or in six hours, if necessary.

In the hemorrhagic form, the single dose is equal to 40,000-50,000 a.u. Later, smaller doses are employed, according to the course of the disease, until the process is reversed.

Babies under one year and between one and two years of age are given a quarter-dose and half-a-dose respectively.

To prevent anaphylactic phenomena, Bezredka's method is recommended whereby 1 ml of serum is injected one or two hours before administration of the full therapeutical dose.

Today, a dialysed, that is protein-free and ferment-processed, serum known as diaferm possessing antitoxin in an extremely high concentration is used for injection of large doses of antitoxin. The seroreaction touched off by this serum is the least possible.

Local treatment for older children consists in antiseptic gargles of dilute boric acid, borax or hydrogen peroxide.

In small children, the mouth is kept clean by spraying or syringing it with the same solutions or 2% baking soda solution.

The most serious complications of diphtheria are lesions of the cardiovascular system (myocarditis) and symptoms of nephrosis. Therefore, all diphtheria patients should be strictly confined to bed and given cardiac stimulants, such as caffeine, camphor, strychnine, etc., as indicated clinically.

Injection of serum is sometimes followed in 7 to 12 days by the onset of serum sickness marked by urticarial rashes, fever and pain in the joints.

The itching is relieved with hot baths, administration of calcium chloride, orally or by intravenous injection, while pain in the joints is allayed with hot-water bottles and hot compresses.

In some cases, three to four weeks after recovery, there is temporary paralysis of the soft palate when liquid food enters the nose and the voice acquires a nasal quality.

Mild forms of such paralysis remit spontaneously, whereas more severe forms are given, where possible, electrical treatment, as well as arsenic and strychnine drugs orally, in *T-rae Strychni* form in doses of as many drops as the child's years twice or three times daily.

Prophylaxis and *nursing care.* The patient with diphtheria should be immediately isolated, preferably in a hospital.

If hospitalisation is delayed, even for a few hours, the patient should be given the first injection of antidiphtheric serum on the spot, since early serum administration is essential for its efficacy.

Anyone having had contact with diphtheria patients must have throat swabs taken for bacteriological examination. They must be isolated and may only leave quarantine seven or ten days later provided all clinical symptoms of diphtheria are absent and the bacteriological tests are negative.

Following his hospitalisation, the patient's flat should be disinfected.

It should be borne in mind that diphtheria bacilli may be discharged not only by active carriers but also by conva-

lescents, who are thus a danger to the community. Their condition is known as the bacilli-carrier state.

Eradication of the bacilli-carrier state is a major problem of diphtheria control. The development of this condition largely depends on the extent of the child's debility following a severe illness as well as on the pathological condition in the nose, pharynx and tonsils, whose treatment must be part of the complex measures against the bacilli-carrier state. Immediate destruction of the bacilli is produced by local, intramuscular or combined administration of antibiotics. The nasal cavity is sprayed with a mixture of penicillin and streptomycin, preferably in the aerosol form, whereas levomycetin, biomycin, tetracycline and erythromycin are given orally in three 0.2 g doses a day for a period of five or six days, etc. Among therapeutical measures, repeated immunisation with diphtheria toxoid is recommended.

Since there is no reliable protection against diphtheria carriers, they must be isolated; in the case of children they may not attend school, etc., and should be kept in the open-air as long as possible, until they are bacilli-free. Antiseptic gargles are also helpful.

The child may be considered free of the bacilli-carrier state only when this has been proved by three examinations of nasal and throat swabs taken consecutively at intervals of a few days.

Diphtheria patients may be discharged from hospital a week after the disappearance of clinical symptoms if the bacteriological tests have proved negative. It is extremely important that information about diphtheria and the measures necessary for its prevention be made widely known among the general public. The ancillary personnel must be taught the rules governing the care of a diphtheria patient; persons in charge of the patient should wash their hands in mercury bichloride solution, 1 : 1,000, after any contact with him. No one should be permitted to sit on the patient's bed, visitors should not be admitted, nor should the patient's utensils be taken from the ward or room. Gauze and cotton wool used to cleanse the patient's nose and mouth must be burned immediately.

The patient's temperature and pulse rate must be carefully watched because weakened heart activity is the most

dangerous symptom in diphtheria. The diet consists of liquid or gruel-like meals of milk, broth, butter and jelly sufficient in vitamins, especially vitamin C, which may be given in ascorbic acid drugs.

CHRONIC INFLAMMATIONS OF THE PHARYNX

Chronic tonsillitis (*tonsillitis chronica*). Chronic inflammation of the faucial tonsils, or chronic tonsillitis, is a frequent disease caused by repeated attacks of acute tonsillitis in the adult. The morbid process is confined to the tonsillar crypts or may primarily affect the tonsillar parenchyma.

The squamous epithelium of the crypts comes off in scales to form fetid caseous masses plugging the crypts and containing numerous bacteria and leukocytes. Owing to the expansion of the crypts the tonsils appear porous and spongy, and the faucial pillars often adhere to the free surface of the tonsils. The crypts become a most convenient place for the retention and propagation of virulent streptococci and staphylococci whose vital activity keeps up the inflammatory process in the tonsils. In unfavourable conditions, like chilling or reduced body resistance, etc., these bacteria may cause exacerbations, such as acute tonsillitis, peritonsillar abscess and a number of general complications, for example, infectious polyarthritis, rheumatic heart, nephritis, etc.

Patients with chronic tonsillitis often complain of discomfort in the tonsillar area, pain on swallowing and, sometimes, of bad breath. There may often be the sensation of a foreign body in the throat and reflex otalgia caused by the pressure of an increased caseous plug. In cases of frequent exacerbations of chronic tonsillitis, the regional lymph glands, both submandibular and cervical, are swollen and tender. Some patients, however, are not conscious of the disease at all.

The patient's history will indicate frequent recurrence of tonsillitis, peritonsillar abscesses, or complications in the form of constitutional diseases immediately consequent upon tonsillitis. Chronic tonsillitis may produce a prolonged slight fever setting in at night for five or six months

and longer as well as a constitutional disturbance manifest in apathy, inability to work, headache, etc.

Consideration of complaints and anamnestic data is not enough for a final judgement; careful examination of both tonsils is necessary to verify the diagnosis.

Treatment. This may be conservative or surgical. The former is by painting the tonsillar crypts with a 5% solution of iodine tincture, trichloracetic acid, 1% Lugol solution or 1-3% silver nitrate solution. If carried out regularly, this procedure frequently causes the tonsils to shrink and stops plug formation. Similar results may be obtained by regular irrigation of the tonsillar crypts with 4% boric acid or penicillin solution, etc. (Fig. 80). Good results are also obtained sometimes by dissection of the tonsillar crypts with a galvanocauter or a blunt-pointed curved knife followed by removal of tissue tags with a conchotome.

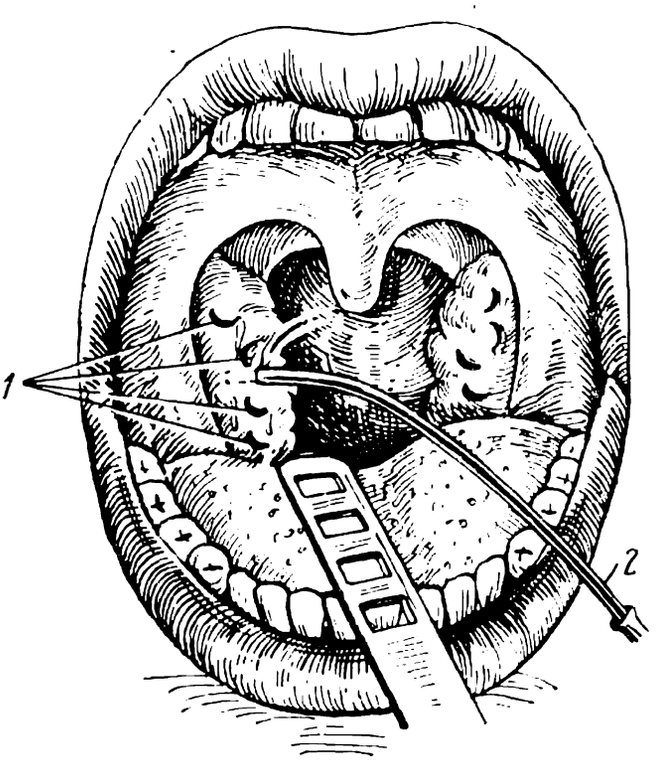


Fig. 80. Irrigation of Tonsil Crypts
(1) tonsil crypts; (2) irrigation cannula

Complete excision of the tonsils is indicated in a frequently recurring peritonsillar abscess and tonsillitis, especially when the latter is liable to cause such complications as rheumatic heart, nephritis, etc. This operation, known as tonsillectomy, comprises the complete enucleation of the tonsils in their capsules (Fig. 81). In nearly all cases, tonsillectomy is a reliable method to prevent relapses of tonsillitis.

The operation is made under local anesthesia at an inpatient clinic. This operation is contraindicated in subjects with a hemopoietic disease or poor blood clotting.

Immediately after the operation, the patient should be carefully watched, because of the likelihood of postoperative

hemorrhages. The patient is forbidden to swallow and instructed to spit saliva into a basin or towel. The patient should refrain from taking food, at least for the first 8 to 12 hours after the operation.

In the early postoperative period, the patient is given a cool and bland diet of milk, broth, jelly, and half-liquid gruels. Naturally, the diet should contain vitamins in plenty.

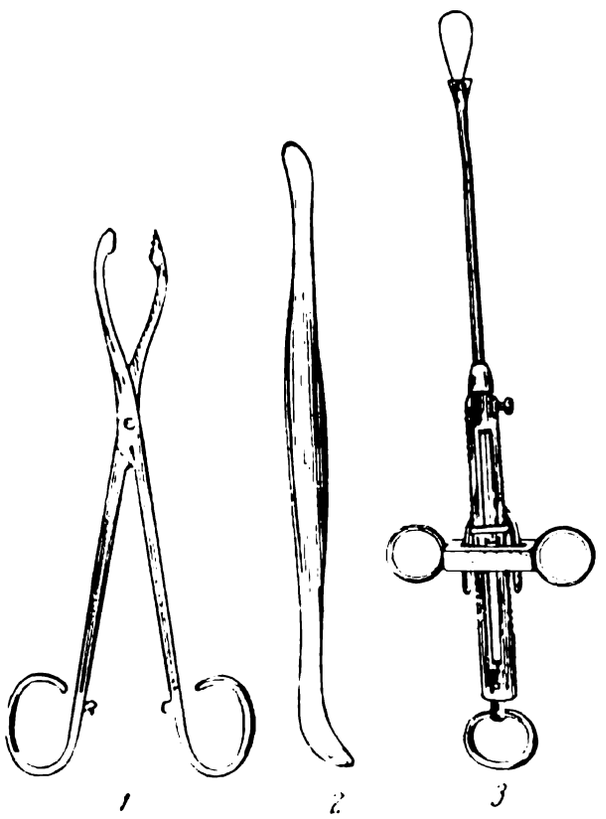


Fig.81. Instruments Used in Tonsillectomy

(1) grasping forceps to engage tonsil; (2) elevator for separation of tonsil; (3) tonsil snare

Chronic pharyngitis (*pharyngitis chronica*). Chronic diseases of the pharyngeal mucosa occur in the catarrhal, hypertrophic and atrophic forms. Their etiology may be of local and systemic character. The former refers to chronic rhinitis, suppuration in a paranasal sinus, chronic tonsillitis, etc. The latter refers to metabolic disturbances and stasis in the venous system in diseases of the heart, lungs, liver and kidneys.

Another important cause is unfavourable climate and occupational hazards presented by dry air, drastic temperature changes and dirty surroundings found in various industries, such as cement plants, porcelain factories, flour mills, etc. Vapour and gases in the chemical industry as well as the constant abuse of tobacco and alcohol may have a pernicious effect on the pharyngeal mucosa.

Symptoms. In the atrophic form of chronic pharyngitis, there is a sensation of tickling, scratchiness and dryness in the throat. In the hypertrophic form, the basic complaint is one of a large accumulation in the nasopharynx of tenacious mucous secretion which causes much coughing and expectoration, especially in the morning, when it may be accompanied by retching and sometimes vomiting.

In the hypertrophic form, the mucosa is red and thickened, and the posterior pharyngeal wall is covered with a sticky mucopurulent secretion which trickles down through the nasopharynx. In the atrophic form, however, the mucosa is dry and glazed as though covered with a thin film of varnish. Sometimes, it is covered with a viscid, almost dry mucus or crusts which are hard to remove even if a medication has been applied.

The lymphoid structures of the mucosa often respond to chronic irritation with a marked hyperplasia of individual follicles exhibited as red granules scattered over the posterior pharyngeal wall and symptomatic of granular pharyngitis. Sometimes there is hypertrophy of adenoid tissue embedded in the lateral pharyngeal folds, which in this case are seen as prominent bright-red and thick bands behind the posterior faucial pillars. This condition is known as lateral pharyngitis.

Treatment. The first essential is to eliminate the basic cause of the condition.

Local treatment of the mucosa comprises the removal of the adherent secretion and alleviation of irritation by frequent throat irrigations with warm alkaline solutions of baking soda, borax and weak, 0.5-1%, saline solutions. For their prescriptions see the section on atrophic rhinitis.

The following gargles are also recommended:

Rp. Natrii benzoici
Natrii biborici
Natrii bicarbonici aa 10.0
M. f. pulv.
DS. Half a teaspoonful in a glass of warm water for a gargle

Rp. Natrii benzoici 3.0
T-rae Opii simplicis gtt. XX
Aq. Amygdalarum amararum 2.0
Glycerini 10.0
Aq. Menthae 50.0
Aq. destill. 200.0
MDS. To be used as a warm gargle three or four times daily, or bicarmint is prescribed in one or two lozenges in half a glass of water for a gargle

In the event of copious secretion, mildly antiseptic and styptic gargles are used two or three times daily. For

this a tablespoonful of camomile or a teaspoonful of sage leaves, which have a stronger styptic effect, are steeped in a glass of boiling water. The tea thus prepared is strained and used as a gargle while being warm.

Warm irrigation has a soothing effect on the mucosa and keeps down hypersensitivity and unpleasant sensations in the throat.

The porous and hypertrophied mucosa is shrunk by painting it with 1%-2%-3%-5% silver nitrate solution once in every two or three days. In the atrophic form, 1% iodine-glycerol or 1% menthol solution in liquid petrolatum are used after a preliminary application of alkaline gargles.

Pharyngomycosis (*pharyngomycosis benigna s. leptothricia*).

This disease is marked by hornification of the squamous epithelium of adenoid tissue. There are white or creamy plaques or thorn-like excrescences found on the unchanged tonsillar surface as well as on the lateral folds of the posterior pharyngeal wall and at the base of the tongue. These pointed or round, as though calcareous, excrescences markedly protruding above the mucosal surface are firmly adherent to their beds. The thorns harbour numerous organisms of the fungus Leptothrix buccalis.

Symptoms. Leptothrix lesions are usually symptomless and are detected only by chance or when they cause a slight pricking sensation. Though this condition is stubborn and resists treatment, it entails no serious complications. Pharyngomycosis is very often mistaken for lacunar tonsillitis.

Treatment. This has been of little effect so far. The common methods are electric cauterisation or regular application to the excrescences of 5% iodine tincture, the use of alkaline gargles with an addition of five drops of iodine in a glass of water, as well as oral iodine in doses of five drops repeated for two or three weeks. Frequently the lesions heal of their own accord.

BENIGN TUMOURS OF THE PHARYNX

Benign pharyngeal tumours which grow as pedunculated papillomas and fibromas from the pillars or soft palate are not dangerous.

Treatment. The tumour is removed by surgery.

Nasopharyngeal fibroma. The nasopharyngeal fibroma is a special type of tumour which occurs almost exclusively in males between the age of 8 to 13 years and in full puberty, i.e., from 20 to 25 years, when the tumour, if still present, begins to shrink. The essential element of a nasopharyngeal fibroma is dense connective tissue containing a great number of elastic fibres and blood capillaries. The tumour is histologically benign, but for its clinical course marked as it is by irresistible growth and destruction of the surrounding tissue, postoperative relapses and frequent copious hemorrhages endangering the patient's life, it may sooner be classified as a malignant neoplasm.

Symptoms. The initial clinical symptom is unilateral nasal obstruction. After four to six months full nasal obstruction occurs as well as more or less marked complications in the ear. In advanced cases with a rapid growth of the tumour it causes the eye, as well as the soft and hard palates to bulge, swells out the nose, etc. Repeated nasal hemorrhages at the very onset of the disease weaken the patient and aggravate his condition still further.

Diagnosis. The tumour is recognised easily. Anterior rhinoscopy usually reveals a red, smooth tumour filling one side of the nose and bleeding readily on probing. When examining the pharynx, a swelling in the soft palate with the edge of a pink, smooth tumour often showing from behind it can be seen. The tumour differs from a choanal polyp by its greater density and broad area of attachment.

Treatment. The treatment is by surgery. The operation is rather difficult because of the deep position of the tumour, its firm adherence to the basal tissue and severe hemorrhage. Small tumours located in the nasopharynx are removed via the nose or mouth. In neglected cases where the tumour invades the zygomatic area and paranasal sinuses the removal is performed only after a preliminary operation has been undertaken to provide access to the tumour proper. This access may be obtained through the antrum of Highmore with complete removal of the lateral nasal wall. Recently surgical diathermy has been successfully used to remove the tumour. Radiotherapy, particularly X-rays, is sometimes also effective.

Cancer of the nasopharynx. This disease is frequently met with after 40 years of age, in younger subjects it is far less frequent. The symptoms of malignant nasopharyngeal tumours should be sought in the progress of the tumour and the functional disturbances attending this progress, particular attention being paid to the initial stages of their development.

According to the primary site of origin the initial symptoms of the tumour may be associated with the following phenomena: (1) otalgia and hearing disorders due to tumour growth beside the Eustachian tube which occurs in six out of every ten cases; (2) nasal obstruction caused by tumour growth in the choanae; (3) neuralgia and paralysis when the tumour grows upwards and presses on the cranio-cerebral nerves. These symptoms are absent in some cases, and the first morbid signs, therefore, may be "lymphadenitis" in the neck and submandibular region caused by metastases into the nearest lymph nodes. Metastases into the cervical lymph nodes are especially early in lymphepithelial tumours.

At first, the patient complains of gradual obstruction of one and then of both sides of the nose. Blowing the nose expels secretion containing blood filaments and sometimes causes nasal hemorrhage or pyoichorous nasal discharge. The patient is usually late in resorting to medical aid. Examination of the nasopharynx by posterior rhinoscopy and palpation with the finger, which is quite indispensable in such cases, will reveal a large or small knobby mass which often disintegrates, bleeds on palpation and merges evenly into the surrounding tissue.

Early diagnosis and correct X-ray treatment of malignant nasopharyngeal tumours improve their outlook, formerly quite hopeless. Recovery may be facilitated by stimulation therapy with repeated blood transfusions in fractional doses, whereas the use of leukopenia-preventive drugs, such as pentoxyl, campolon, etc., permits an adequate dose of irradiation to be administered to cases which until recently were regarded as incurable.

Malignant tumours in the middle and lower pharynx. A malignant tumour in the form of a dense knobby infil-

tration, which very easily ulcerates due to its traumatic irritation by the passage of food, may develop on the soft palate, tonsils (Fig. 82), posterior and lateral walls of the pharynx or in its lower portions, namely, the pyriform sinuses. With the onset of ulceration in the tumour, it begins

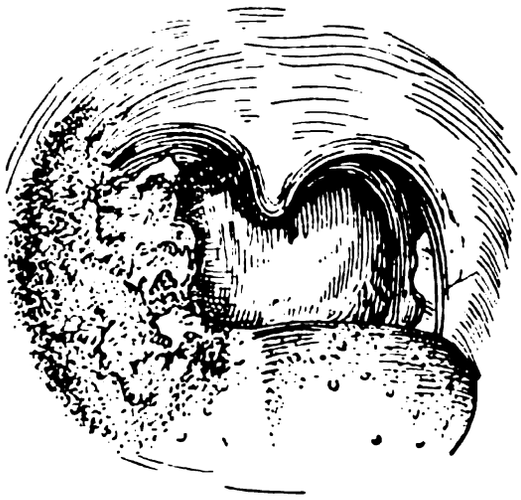


Fig. 82. Cancer of tonsil

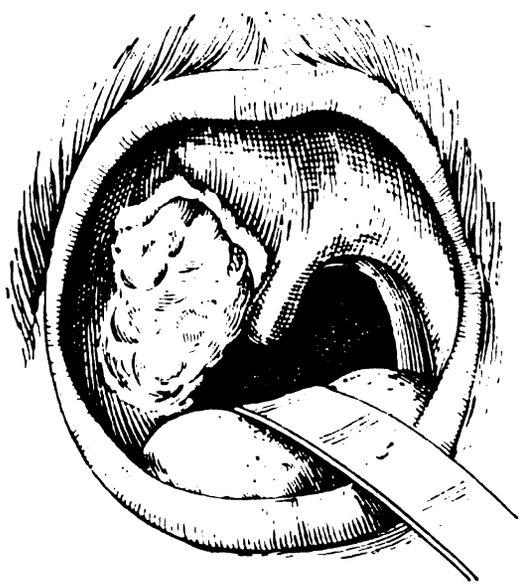


Fig. 83. Sarcoma of tonsil

to ache, grows in size and interferes with the passage of food. There are early metastases to the nearest lymph glands in the neck and at the base of the skull.

Surgical removal of these tumours from healthy tissue is often belated and its outcome may, therefore, be unfavourable. New hope has been given by X-ray and radium or radio cobalt therapy after a preliminary ligation of the external carotid artery. This treatment sometimes arrests the growth of the tumour for a long time and so delays the fatal outcome.

Lymphosarcoma of the tonsils (Fig. 83) occurs more frequently than other kinds of *pharyngeal sarcoma*, and mostly in young people.

DISEASES OF THE LARYNX

ANATOMY OF THE LARYNX

The larynx is a part of the wind-pipe, the upper end of which opens into the pharynx, through which it communicates with the oral and nasal cavities, while its lower end continues as the lumen of the trachea. The larynx lies in the anterior portion of the neck on a level with the fourth

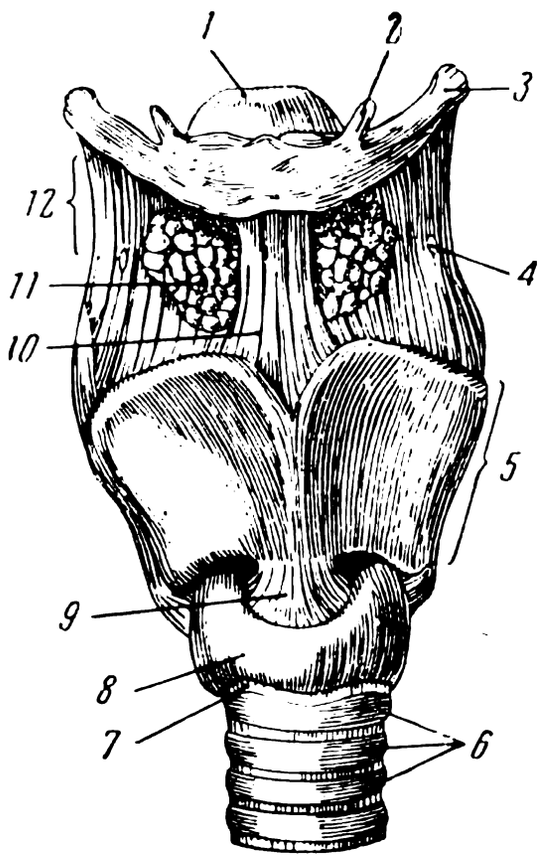


Fig. 84. Ligaments and Joints of Larynx (Front View)

(1) epiglottis; (2) lesser cornu of hyoid bone; (3) greater cornu of hyoid bone; (4) opening in thyrohyoid membrane; for passage of superior laryngeal nerve; (5) thyroid cartilage; (6) tracheal cartilages; (7) cricotracheal ligament; (8) cricoid cartilage; (9) *conus elasticus*; (10) middle thyrohyoid ligament; (11) *corpus adiposum*; (12) thyrohyoid membrane

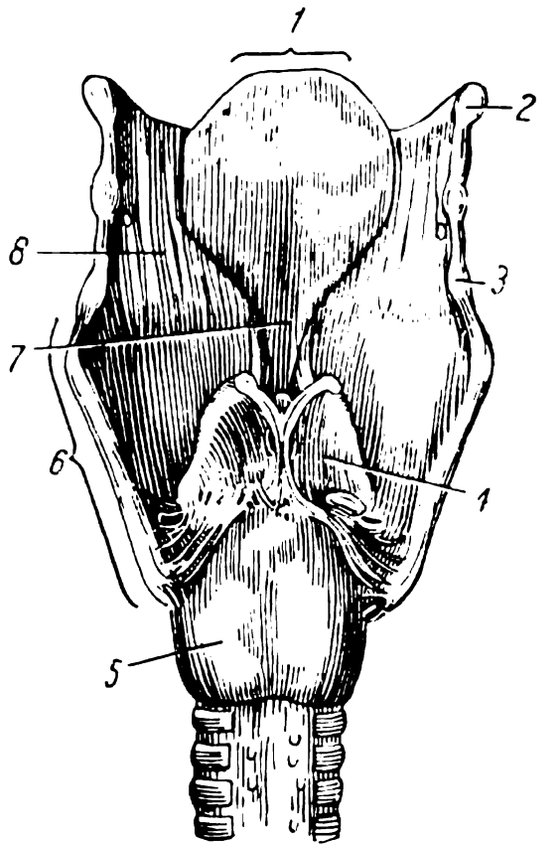


Fig. 85. Ligaments and Joints of Larynx (Rear View)

(1) epiglottis; (2) greater cornu of hyoid bone; (3) superior cornu of thyroid cartilage; (4) arytenoid cartilage; (5) cricoid cartilage; (6) thyroid cartilage; (7) *petiolus* of epiglottis; (8) thyrohyoid membrane

and sixth cervical vertebrae. A broad ligament connects the larynx with the hyoid bone above.

The laryngeal skeleton is made of cartilage (Figs. 84 and 85). The basic cartilage is the cricoid which resembles a signet ring in shape. Its narrow part faces outwards, while its broad, so-called signet portion looks backwards. Above it lies the thyroid cartilage which consists of two wings or alae joined together at an angle and forming a notch at their junction. As this portion of the thyroid cartilage is covered with skin alone, it may be easily felt with the fingers, and in men it protrudes at the front of the neck, and is known as "Adam's apple". On the upper surface of the posterior part of the cricoid there are two arytenoid cartilages with two processes at their base, namely, the muscular and the vocal. The vocal muscle is attached to the latter. In addition, the laryngeal aperture is covered by a special cartilage, known as the epiglottis, which is attached by ligaments to the upper margin of the notch in the thyroid cartilage. All the laryngeal cartilages are bound together by numerous ligaments as well as by their joints.

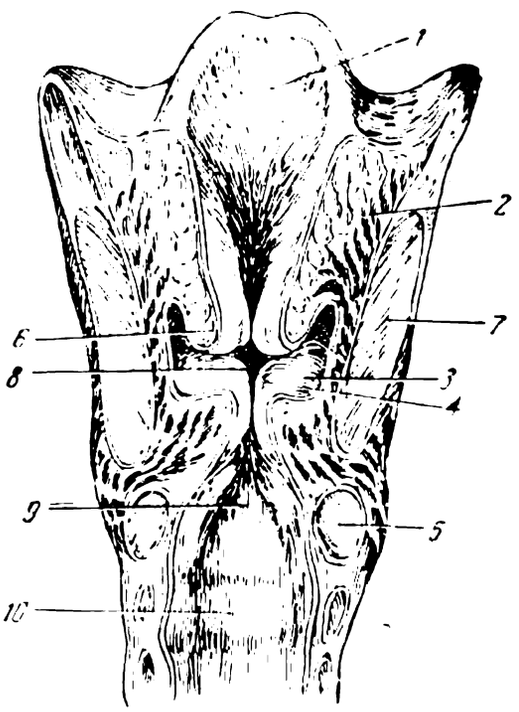


Fig. 86. Larynx in Vertical Section

(1) epiglottis; (2) extrinsic thyroarytenoid muscle; (3) Morgagni's ventricle; (4) true vocal cord; (5) cricoid cartilage; (6) false ligament; (7) thyroid cartilage; (8) glottis; (9) subglottic region; (10) trachea

The true vocal cords comprising the paired vocal, or thyroarytenoid, muscle are prominent in the laryngeal space. The vocal cords are stretched between the inner surface of the thyroid cartilage and the vocal process of the arytenoid cartilage on the appropriate side. In respiration, the vocal cords form a triangular opening for the passage of air known as the rima glottidis or the glottis (Fig. 86). In phonation the vocal cords draw nearer together.

Above the true vocal cords lie the false vocal cords which are simply two folds of mucosa. Between the true and

the false cords, on the sides, there are two slit-like pockets, the so-called Morgagni's ventricles whose mucosa has numerous glands which moisten the vocal cords.

The laryngeal muscles. These may be divided into the extrinsic and the intrinsic muscles. The former connect the larynx with other parts of the skeleton. They lift and lower the larynx, or fix it in a certain position.

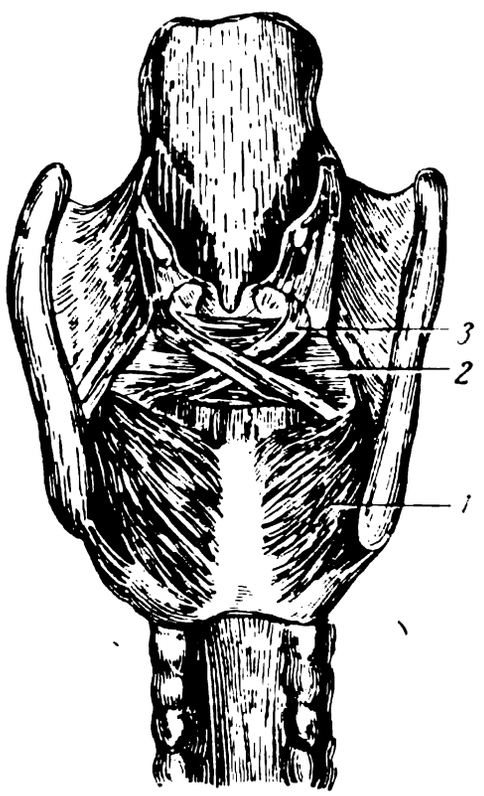


Fig. 87. Laryngeal Muscles

(1) posterior cricoarytenoid muscle; (2) transverse interarytenoid muscle; (3) oblique interarytenoid muscles

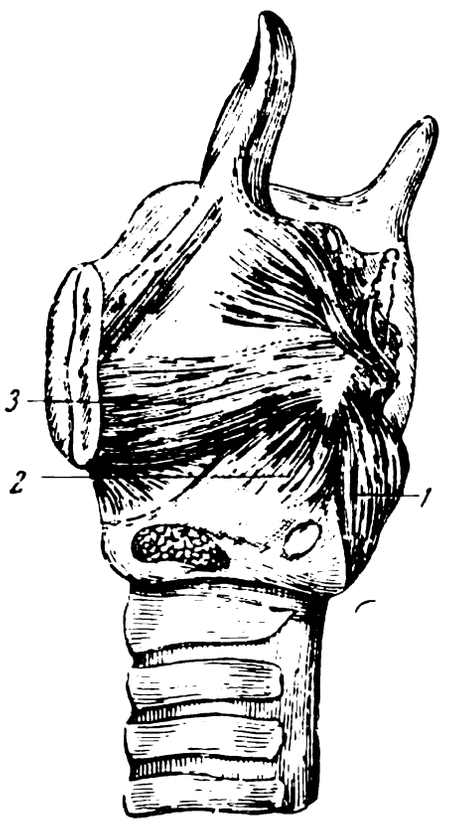


Fig. 88. Laryngeal Muscles

(1) posterior cricoarytenoid muscle; (2) lateral cricoarytenoid muscle; (3) intrinsic thyroarytenoid muscle

The intrinsic muscles are attached to the inner and outer surfaces of the larynx and do not extend beyond its limits. It is these muscles that perform the laryngeal functions of respiration and voice production. In accordance with these functions, the intrinsic laryngeal muscles divide into the constrictors and the dilators of the glottis. The basic, respiratory function of the larynx is performed by one paired muscle, namely, the posterior cricoarytenoid muscle, or simply the posterior muscle (Fig. 87), the only muscle which dilates the glottis; all the other muscles directly or

indirectly serve to close the glottis. The antagonist of the posterior cricoarytenoid muscle is the lateral cricoarytenoid muscle (Fig. 88) which draws the vocal cords together and, consequently, narrows the glottis. The interarytenoid muscles, the transverse and the oblique, bring the arytenoid cartilages together and close the posterior part of the glottis. The vocal cords are kept in tension by the above-mentioned vocal, or thyroarytenoid muscle. The anterior cricothyroid muscle tenses the vocal cords, since it is attached to the cricoid and thyroid cartilages and in contraction lengthens the larynx in the anteroposterior aspect. The laryngeal mucosa is composed of elastic fibres and covered with a ciliated columnar epithelium, excluding the true vocal cords, aryteno-epiglottic ligaments, laryngeal surface of the arytenoid cartilages and interarytenoid space covered with a stratified squamous epithelium.

The larynx is innervated by two branches of the vagus nerve, namely, the superior and the inferior laryngeal nerves. The former is primarily a sensory nerve which supplies sensation to the entire laryngeal mucosa. This nerve also has one motor branch extending to the anterior cricoarytenoid muscle. The nerve supply to all other laryngeal muscles is through branches of the inferior laryngeal nerve.

The larynx is not only organ for the passage of air but also for voice production. The sound is produced by the vibration of tense vocal cords during the passage of air in expiration through the glottis closed to a narrow chink.

METHODS OF EXAMINING THE LARYNX

Examination of the larynx is conducted by means of *direct visual inspection* or *mirror laryngoscopy* with a laryngeal mirror attached to the handle of another laryngeal instrument (Fig. 89).

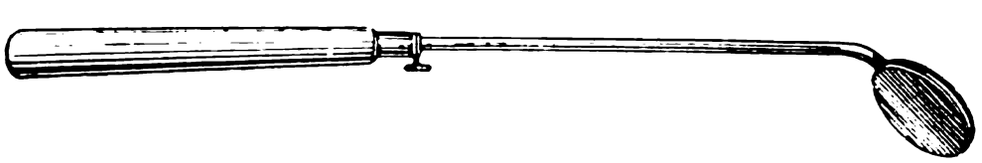


Fig 89. Laryngeal Mirror with Handle

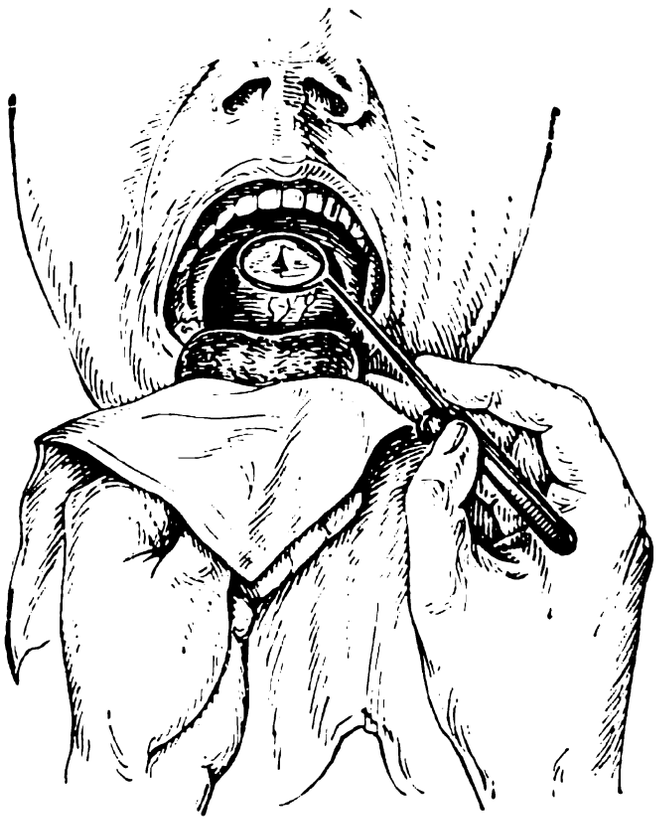


Fig. 90. Mirror Laryngoscopy

In laryngeal examination the patient is asked to lean slightly forward and to put out his tongue which is then held in position with a square gauze throughout the remainder of the examination. The mirror is slightly warmed, glass side down, in the flame of an alcohol lamp or in hot water, so that it will not fog on the patient's breath when inserted in the mouth.

The laryngeal mirror is placed in the mouth, mirror side down, at an angle of 45° to the horizontal, with its back surface pushing the uvula and soft palate backwards and upwards, care being taken not to touch the posterior pharyngeal wall to avoid the gagging reflex. At the same time, the patient is told to breathe quietly and drawl the sound "e-e-e-e-e-e". This raises the epiglottis and the larynx can be viewed reflected in the mirror (coloured Table IV, Fig. 1). The epiglottis is the first organ visible and by changing the position of the mirror a thorough examination of all its parts can be made, after which the vocal cords and interarytenoid region are subjected to the same scrutiny. Examination of the larynx can be considered successful only when all of its parts have been inspected, and mobility of the vocal cords in quiet breathing and phonation (voice production) has been ascertained (Fig. 90). It should be remembered that in mirror laryngoscopy all anterior parts of the larynx will appear as if being above, and all the rear parts will be seen as if below. The lateral parts will not change their positions in mirror reflection (Fig. 91, a, b).

The larynx can also be examined by means of *direct laryngoscopy*, in which inspection is made without a mirror.

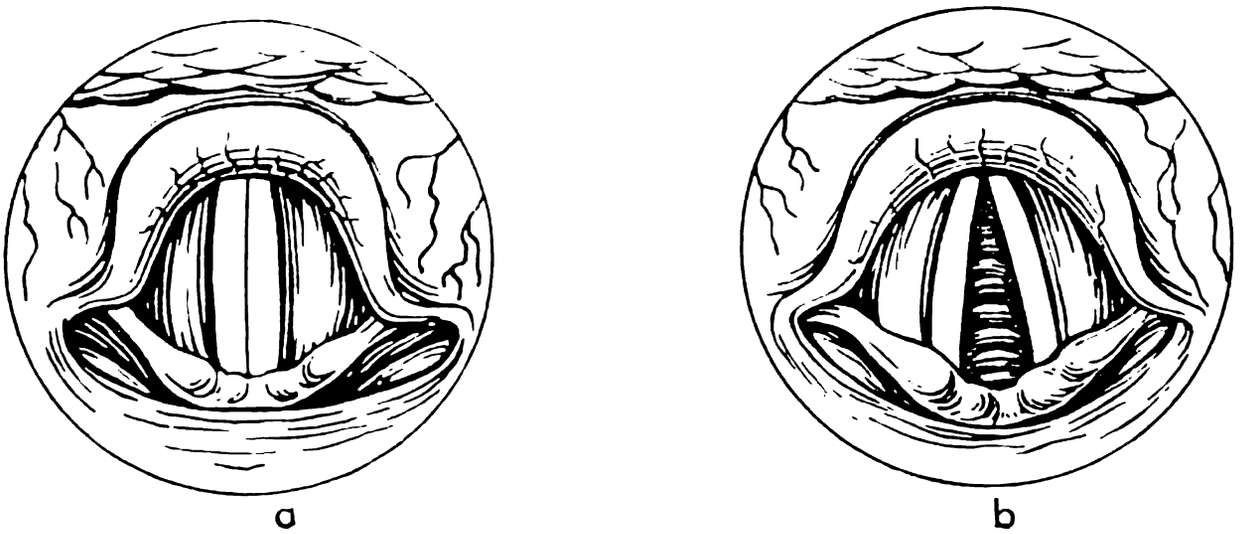


Fig. 91. Mirror View of Larynx
(a) in phonation; (b) in respiration

An angular spatula is firmly pressed against the back of the tongue to push it forward. The patient's head is gradually tilted backwards to bring the oral cavity, pharynx and larynx in one straight line. In this position, the epiglottis, arytenoid cartilages and vocal cords may be examined in turn with the aid of an appropriate light. In modern practice, the angular spatula has been supplanted by a more convenient instrument called the laryngoscope. Laryngeal mirrors marked with the letter "K" on the reverse side may be kept in boiling water. Other types of mirror cannot resist boiling and are sterilised in antiseptic solutions of lysol or carbolic acid, washed in boiled water and wiped dry with a square of gauze.

GENERAL METHODS OF TREATMENT IN LARYNGEAL DISEASES

The larynx is *painted* or *lubricated* with the aid of threaded *laryngeal probes* made of soft metal, in order that the probe may be curved appropriately. Cotton wool should be firmly wrapped on the probe to prevent it slipping off in lubrication with untoward results.

All manipulations within the larynx, including lubrication, are made under the guidance of vision aided by a laryngeal mirror. As soon as the laryngeal probe topped with a drug-soaked cotton tuft is reflected in the mirror,

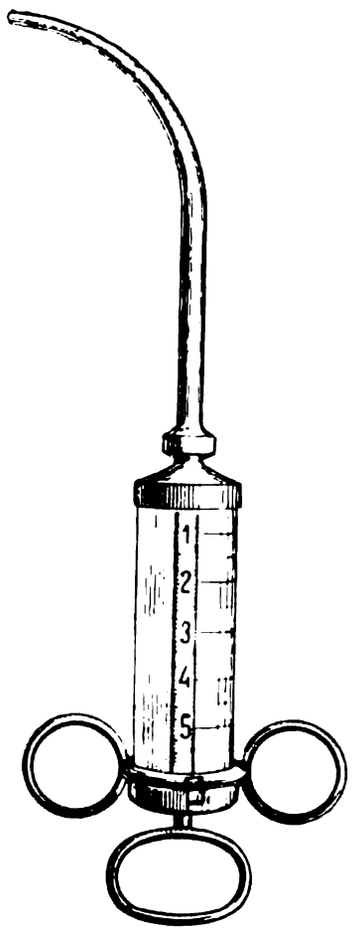


Fig. 92. Laryngeal Syringe

it is quickly pushed behind the epiglottis into the larynx. Contact between the probe and the vocal cords will produce coughing, and in some cases a laryngeal spasm. Therefore, before lubrication, the patient should be warned of the possibility of this spasm as well as of its harmlessness. To stop the spasm quickly, the patient is directed to hold his breath for a few seconds, and then take a deep breath with his mouth wide-open.

A metal-tipped *laryngeal syringe* (Fig. 92) is used for pouring medicinal solutions into the larynx as well as for its anesthesia. The tip must be boiled before every application. A small amount of medicine is drawn into the syringe and instilled into the larynx in a 0.5-1 mg dose. This is a more delicate procedure than laryngeal lubrication and seldom causes a laryngospasm.

Insufflation of powder into the larynx is made with the same type of insufflator as is used for the nose, but it has a curved beak to fit the shape of all other laryngeal instruments. A small amount of the powder is put in the insufflator. As in all other manipulations, insufflation is performed under the guidance of vision. Compression of the bulb often displaces the beak at the moment of insufflation so that the powder is blown into the pharynx. With adequate skill, however, gained from experience in laryngoscopy, the powder will always be discharged in the right direction. The dust should not be blown into the larynx in large quantities for fear of a laryngospasm.

Laryngeal diseases are treated by inhalation of various drugs pulverised with *cold* and *vapour atomisers.*

A special apparatus known as the inhaler is used for steam inhalation (Fig. 93) and has the following components: (1) a water boiler heated with an alcohol lamp or by electricity; (2) a metal tube bent at right angles

and connected with the water boiler, and (3) a second tube whose one end is in a glass with liquid medicine and the other, narrow end closely adjoins the horizontal section of the first tube. The vapour formed in the boiler enters the first tube, draws the solution from the glass through the second tube and finally escapes through a wide glass tube facing the patient. The latter inhales the finely

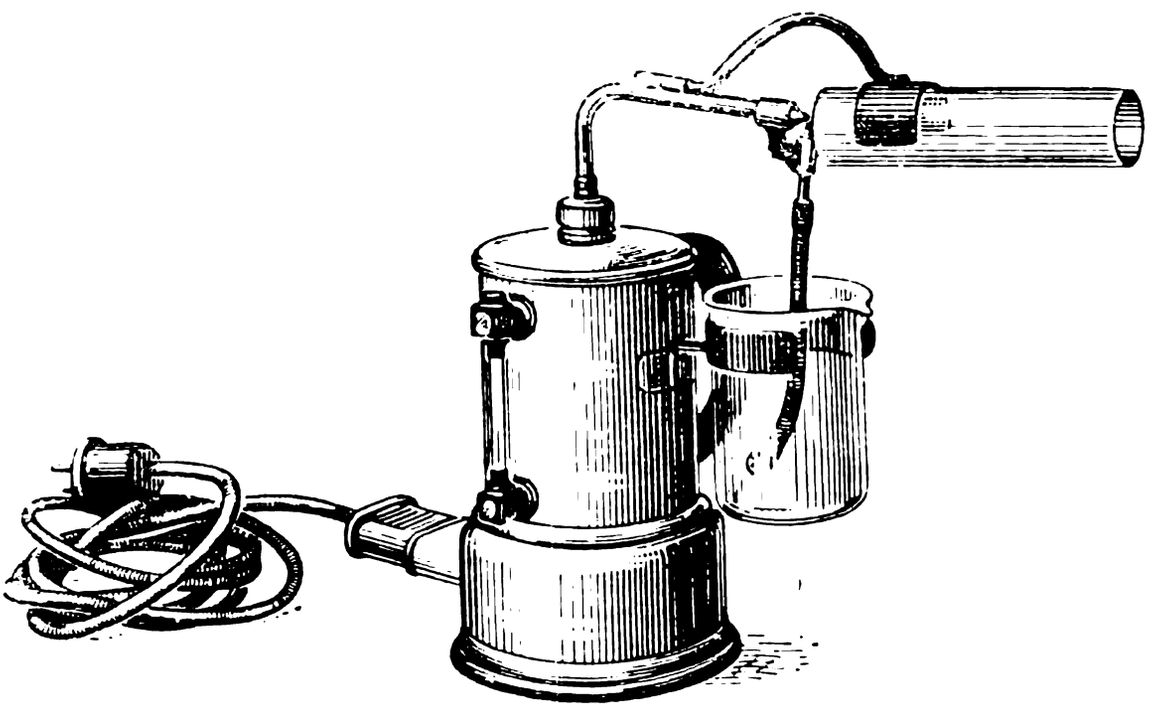


Fig. 93. Electric Vaporiser

dispersed medicinal substance contained as tiny drops in the warm vapour.

Medicinal sprays of the larynx, as well as of the naso-pharynx and pharynx, can be made with appropriate atomisers. The atomiser (Fig. 94) consists of a plugged bottle containing a slender metal tube, whose lower end comes down near the bottom, while the upper end is arched and equipped with a sieve-like plate. The first tube is joined at right angles by another tube connected with two coupled rubber bulbs. Compression of the first bulb with an opening for air suction and a valve to keep air from free escape pushes the air into the second bulb which by contraction supplies a continuous air stream to the external end of the tube. This stream draws up liquid from the bottle and shoots it forth in a fine spray. The patient is told to insert the

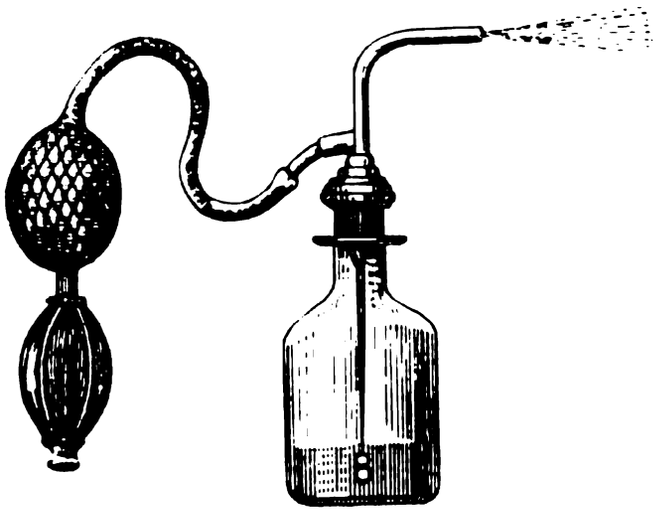


Fig. 94. Atomiser

upper end of the spray tube into his mouth, but not too deeply, so that gagging may be avoided. At the same time, the bottle is held in the left hand, and the rubber bulb is compressed by the right hand 20 to 25 times every session. Both warm solutions and those at room temperature are employed as sprays. In some cases, oily solutions are applied to prevent quick drying of the mucous membrane, etc. Special type of sprayers are then used. In view of its anatomical structure, all instruments inserted into the larynx are bent appropriately to fit its shape. Before each insertion, the instruments must be checked to see that they are in good order, so that there is no danger of any part accidentally slipping into the trachea and bronchi.

GENERAL SYMPTOMS OF LARYNGEAL DISEASES

The most dangerous symptoms of a laryngeal disease are *respiratory disturbances* caused by constriction of the glottis in the case of laryngeal edema, diphtheria, tuberculosis, foreign bodies, tumours, bilateral paralysis of the dilator muscles, and other diseases. Respiratory obstruction may be mild, almost symptomless, or severe, with symptoms of short breath, cyanosis and cardiac disorder.

The most common symptom of a laryngeal disease is either a *changed voice* which becomes more or less hoarse, or complete aphonia. Such symptoms are found in all laryn geal diseases impairing the mobility and correct configuration of the vocal cords.

Pain may be felt within the larynx or on its outer surface. Intralaryngeal pain which often radiates to the ear is caused by phlegmons and ulcerative processes, perichondritis and sharp foreign bodies. The most severe pain is produced by ulceration of the arytenoid cartilages and the external

surface of the epiglottis. In acute laryngitis, there is a sensation of scratchiness, tickling and burning in the throat which may cause coughing.

Laryngeal cough may be wet or dry, usually occurring at the onset of an acute inflammation or in dry chronic catarrh. The inflammation and swelling of the subglottic area and trachea are accompanied by a dry, harsh cough.

ACUTE LARYNGITIS
(*Laryngitis acuta*)

Acute laryngitis is most commonly found in acute catarrh of the upper respiratory tract. The disease is often associated with total or partial chilling of the body. It may also be caused by inhalation of acrid vapours, very dirty air, as well as by overexertion of the vocal cords, such as in prolonged and loud talking. Laryngeal inflammation may also be the result of highly contagious diseases, such as measles, scarlet fever and typhoid.

Course and *symptoms.* Laryngoscopy reveals diffuse hyperemia of the laryngeal mucosa, more or less markedly swollen and congested true vocal cords, and clots of viscid secretion. Phonation sometimes fails to produce complete approximation of the vocal cords due to paresis of the vocal muscles. According to the degree of the laryngeal lesion the voice becomes hoarse and rough with cough, dry at first and accompanied by a feeling of dryness and burning in the throat. The scanty sputum, which is hard to expectorate, gradually grows in quantity, whereupon it may be expectorated with ease producing a marked amelioration in the patient's general condition.

The general feeling of illness is sometimes combined with headache and a slight fever. The disease commonly persists for only seven to ten days and passes quickly if treatment is adequate.

Treatment. The basic remedy is to remove the harmful factors which have provoked the onset of the disease. The patient must give his larynx complete rest by speaking as little as possible for five to ten days depending on the severity of the case. He is advised at first to inhale penicil-

lin aerosols and then proceed with alkaline inhalations a few times daily and a hot compress on the neck.

The following prescriptions may also be recommended:

Rp. Cocaini hydrochlorici 0.1
Aq. Amygdalarum amararum 4.0
Glycerini puri 6.0
Aq. Menthae 50.0
Aq. destill. 150.0
MDS. For steam inhalation

If a vaporiser is not available, the following prescription may be used for inhalation:

Rp. Mentholi crystallisati 1.0
Spiritus vini rectificati 10.0
MDS. Ten to twenty drops in a glass of boiling water for steam inhalation

Febrifuges and narcotics, such as codeine powder, are given for fever and cough, and mixtures are used to promote expectoration.

Codeine or dionin may be used in drops or solution together with bromine drugs.

Rp. Codeini puri 0.15
Aq. Amygdalarum amararum 15.0
MDS. Ten to fifteen drops two or three times daily

Rp. Dionini 0.3
Aq. Laurocerasi 15.0
MDS. Fifteen to twenty drops three times daily

Rp. Ammonii bromati 4.0-8.0
Codeini puri 0.15
Sirupi simplicis 8.0
Aq. destill. 180.0
MDS. One tablespoonful three or four times daily

In case of marked dryness of the mucosa and dry crusting, oily solutions are applied to the larynx, such as:

Rp. Chloretoni 0.5
Mentholi crystallisati 1.0
Ol. olivari 50.0
MDS. For laryngeal infusion

Frequent ventilation of the room and clean fresh air assist speedy recovery.

Prophylaxis. See section on prevention of diseases of the upper respiratory tract (pp. 135-136).

CHRONIC LARYNGITIS
(Laryngitis chronica)

Chronic laryngitis follows repeated acute attacks. Furthermore, chronic laryngeal inflammation may be due to irremovable factors which cause acute laryngeal catarrh present in habitual alcoholics and inveterate smokers. Occupational laryngeal catarrh is often met with among singers, teachers and people who work in dusty surroundings or are exposed to chemical hazards. Long-continued inflammations in the upper airways, such as chronic coryza, paranasal sinus suppuration, tonsillitis, pharyngitis, etc., as well as in the lower portions of the respiratory tract, such as tracheitis, purulent bronchitis, may cause a chronic inflammation of the larynx.

Symptoms. These are similar to those of acute laryngitis, though somewhat milder. At times, there is exacerbation and aggravation of the condition in the affected area. The patient complains of hoarseness which grows worse at times, quick onset of vocal fatigue, as well as of tickling and scratchiness in the throat with a constant temptation to cough.

The laryngeal mirror shows that the laryngeal mucosa is of a grey red colour and in some places swollen, particularly along the margins of the true vocal cords and in the interarytenoid area. The mucous membrane of the false vocal cords may be edematous and swollen and sometimes partially covers the true vocal cords, thus interfering with phonation. Small patches of cornifying epithelium, known as *pachydermia laryngis*, most commonly develop in areas covered with squamous epithelium, i. e., the area of the true vocal cords and the interarytenoid area. Small edematous thickenings of the mucosa, known as *singers' nodules* (Fig. 95), sometimes form on the vocal cords in front of each other. They interfere with phonation and cause hoarseness. Atrophy of the mucous membrane sometimes entails the formation of

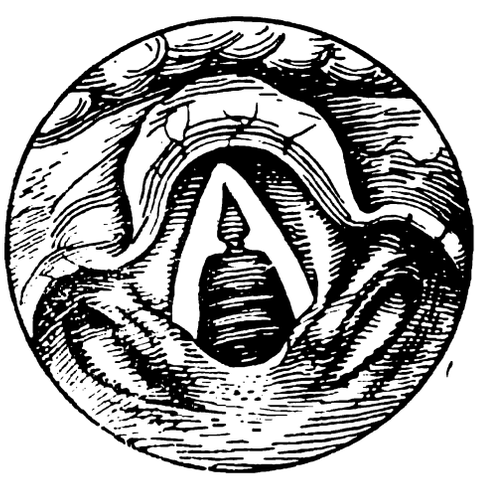


Fig. 95. Nodules on Vocal Cords

crusts which cause an agonising cough. The atrophic form of laryngitis is commonly attended with atrophic catarrh of the pharynx and nose.

Treatment. It is necessary to remove all predisposing and underlying causes of catarrh becoming chronic by refraining from smoking, strong drinks and overstraining the voice. Treatment of the nose and nasopharynx should be undertaken simultaneously since oral respiration is harmful to the larynx. The laryngeal mucosa is painted every other day with 1%-2%-3% silver nitrate or 1% tannin solutions prepared by the following formula:

Rp. Sol. Argenti nitrici 1-2-3% 10.0
MDS. For laryngeal painting

Rp. Tannini 1.0
Glycerini 10.0
MDS. For laryngeal painting

Inhalation of alkaline vapours of baking soda and natrium benzoicum solutions are used, as well as iodine-glycerol given for atrophy and crusting.

Apart from painting the larynx with iodine-glycerol, infusions of an aqueous iodine-glycerol solution are also helpful, and their effect is somewhat milder. The formula of this solution is as follows:

Rp. Jodi puri 0.15
Kalii jodati 1.0
Aq. destill. —
Aq. Menthae aa 100.0
Glycerini 10.0
MDS. For laryngeal infusion

Inhalations are given a few times daily with special instruments described above. If these are not available, two or three glassfuls of boiling water should be poured into a basin and the steam inhaled through a make-shift paper tube for two or three minutes a few times daily. Penicillin is an effectual remedy against exacerbations of chronic laryngitis.

Prophylaxis. To ward off this disease, one should avoid a sudden exposure of the larynx to cold upon leaving a heated room, abstain from singing and loud talk in the frost after a hot bath as well as from continual straining of the voice in dry and dusty surroundings, particularly in the absence of nasal breathing.

LARYNGEAL PERICHONDRITIS
(*Perichondritis laryngis*)

Perichondrial inflammation of the laryngeal cartilages may be primary due to trauma or secondary due to infectious diseases both acute (typhoids and child infections) and chronic (tuberculosis or syphilis) as well as to malignant new growths.

Symptoms. In the acute form there is pain on swallowing and speaking and a low-grade fever. Laryngoscopy reveals marked edema of the inflamed areas, which invades the adjacent regions of the mucosa and causes a severe stricture of the larynx. Respiratory obstruction in cases of a rapidly developing edema may turn into asphyxia and require surgical interference, i.e., tracheotomy. Other cases may produce abscesses with partial or complete necrosis of cartilage and formation of fistulas. The progress of inflammation involves cicatrisation, which leads to chronic laryngeal stenosis and jamming of the tracheotomy tube.

Treatment. In acute cases, the symptoms of inflammation should be relieved by rest in bed as well as by ice to suck and cold compresses applied to the neck. If symptoms of imminent asphyxia appear tracheotomy is imperative. In cases of chronic cicatricial stenosis of the larynx the patency of its lumen is restored by surgery.

BENIGN TUMOURS OF THE LARYNX

Benign laryngeal neoplasms commonly occur as fibromas and papillomas and less frequently as angioma and cysts.

Fibroma (Fig. 96) is a small tumour varying in size from a pin-head to a pea, which most commonly develops in the anterior and middle thirds of the true vocal cords. The tumour is a pale-rosy or pink smooth mass attached to a pedicle or a fairly broad base. It is frequently caused by chronic irritation of the mucosa.

Papilloma (coloured Table IV, Fig. 2) is a multiple greyish-red tumour appearing as a warty and clustery growth resembling a cauliflower or a cock's comb. It may originate

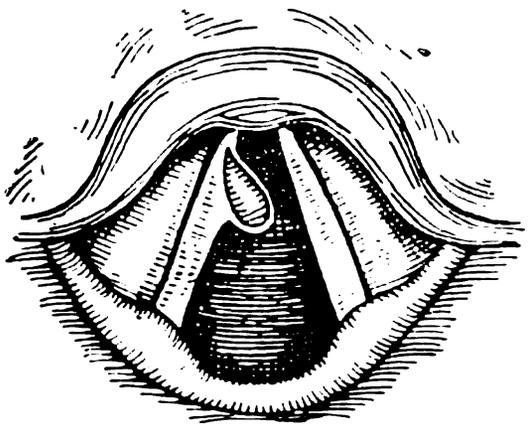


Fig. 96.
Laryngeal Fibroma

in the true vocal cords, and also from any place of the glottis and epiglottis.

In the majority of cases, this tumour appears in children aged 2-8-10 years and owing to its speedy growth fills the entire glottis in a brief space of time.

Angioma is a small and soft tumour with a rough surface and a characteristically purple colour.

Small *cystose tumours* encountered in the larynx develop due to the closure of mucous gland outlets and resemble mucous nasal polyps.

Symptoms. The common symptom of a benign tumour is hoarseness. Aphonia usually sets in gradually rather than suddenly and in case of the tumour being located above or below the glottis, the voice is lost only when the tumour is pressed between the vocal cords. The quick growth of papillomatous tumours, in children in particular, often leads to complete aphonia and obstruction of breathing, necessitating tracheotomy.

Treatment. Conservative treatment is not very effective. Therefore, surgery is the principal treatment for benign laryngeal tumours which are removed with a laryngeal forceps in what is known as the endolaryngeal operation (Figs. 97 and 98). Papillomatous new growths in the larynx of children are removed by the endolaryngeal method, whereas

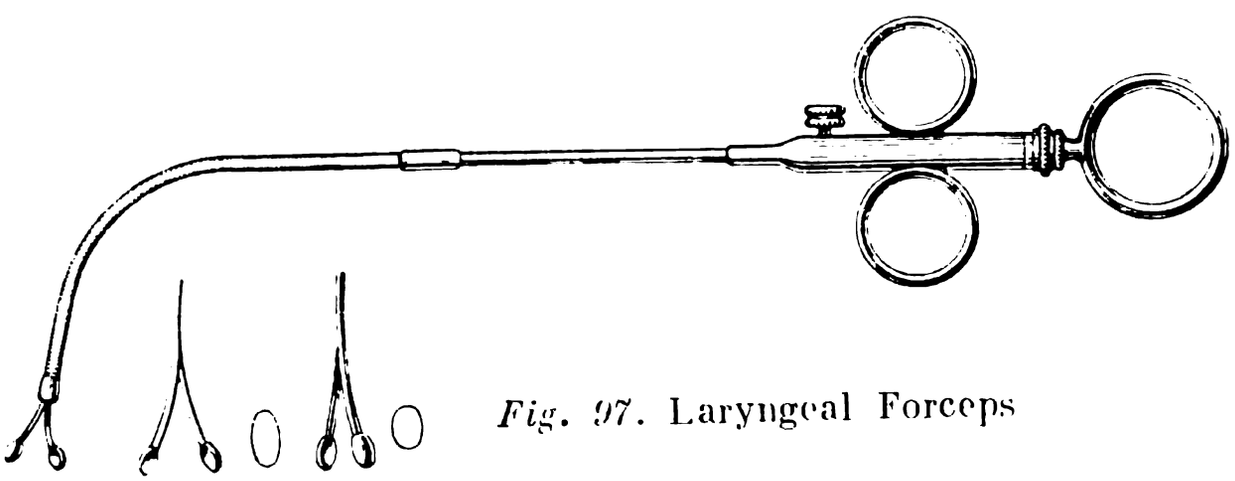


Fig. 97. Laryngeal Forceps

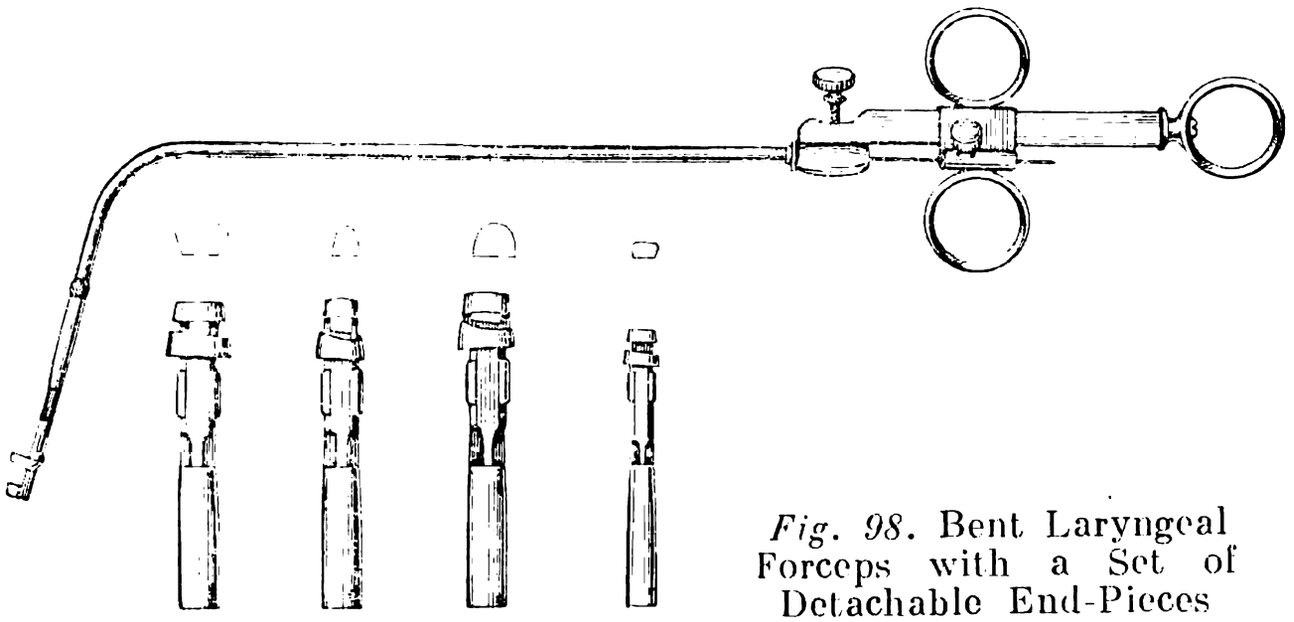


Fig. 98. Bent Laryngeal Forceps with a Set of Detachable End-Pieces

the more complicated cases may require external excision of the larynx, otherwise known as laryngofissure. The excision of the tumour is followed by X-ray therapy to prevent a recurrence.

MALIGNANT TUMOURS OF THE LARYNX

Laryngeal cancer (*cancer laryngis*). This condition commonly occurs after 40 years of age, primarily in smokers, although it can occur in younger people. The early symptoms of laryngeal cancer are congestion and infiltration in one vocal cord as well as a limited and usually unilateral fixation of the vocal cords and slight hoarseness which does not respond to conservative treatment.

The tumour growth is of gradual onset. At first, there is a hardly noticeable slight congestion and infiltration inconspicuously invading the surrounding tissues. This condition may continue for months.

In more advanced cases, in addition to chronic laryngitis a tumour appears as an immobile dense infiltrate on one half of the larynx (coloured Table IV, Fig. 3). In this stage, the hoarseness becomes worse or complete aphonia sets in.

Some parts of the tumour may ulcerate, and the decomposing tissue will cause fetor, harsh cough and pain on swallowing which occurs very early in ulceration of the arytenoid cartilages, aryepiglottic folds, and epiglottis proper.

The nearest lymph nodes are swollen and adherent to the skin. The patient dies of secondary pneumonia, asphyxia or hemorrhage caused by the growth or decay of the tumour.

Diagnosis. It is sometimes very difficult to identify laryngeal cancer, particularly in its early stages. In doubtful cases, the diagnosis should be verified by biopsy, inasmuch as surgical interference and radiotherapy can save the patient only at an early stage of the disease.

Prognosis. The forecast for laryngeal cancer is poor, particularly if the diagnosis has been delayed, and especially if the perilaryngeal lymphatic nodes have been involved in the process. The prognosis at an early stage of the disease is more favourable.

Treatment. The only hope lies in the earliest possible surgical interference or X-ray and radium therapy of the larynx. A common operation today is to remove one half or the whole of the larynx. More limited excision may be successful only at the initial stages of cancer.

Sarcoma of the larynx (*sarcoma laryngis*). This disease is quite rare. Externally it resembles a laryngeal fibroma and, sometimes, angioma.

It is more common in childhood, and its quick development entails an equally quick respiratory obstruction.

The outlook is usually poor. Treatment may be both surgical and conservative, the latter with X-rays, radium, etc. Relapses, however, are frequent with either form of treatment.

ACUTE AND CHRONIC STENOSES OF THE LARYNX

Acute stenosis of the larynx is a quick and sometimes sudden narrowing of its lumen, which, if considerable, can cause asphyxia.

The stricture of the glottis may be caused by inflammatory changes in the larynx, a sudden spasm of the glottis, a lesion of the laryngeal motor nerves or may result from a trauma.

Laryngeal inflammations in adults involve considerable breathing difficulties in cases of edema of the mucous membrane in separate parts of the larynx, commonly the epiglottis and arytenoid cartilages (coloured Table IV, Fig. 4).

Such swellings are due to various infectious diseases, erysipelas, laryngeal phlegmon, influenza, as well as to traumas and foreign bodies. The dyspnea developing in laryngeal stenosis is characterised by stridor and difficult inspiration, as distinct from stenosis of the low-lying bronchi where exhalation is usually obstructed. At first, the oxygen shortage is compensated for by a slower and deeper respiration; in continued stenosis the larynx starts to push up and down with every single breath, while every inspiration causes the intercostal and supraclavicular spaces to be drawn deeply inside. Later on, the ominous signs of asphyxia appear, that is cyanosis, slow and shallow respiration and loss of consciousness.

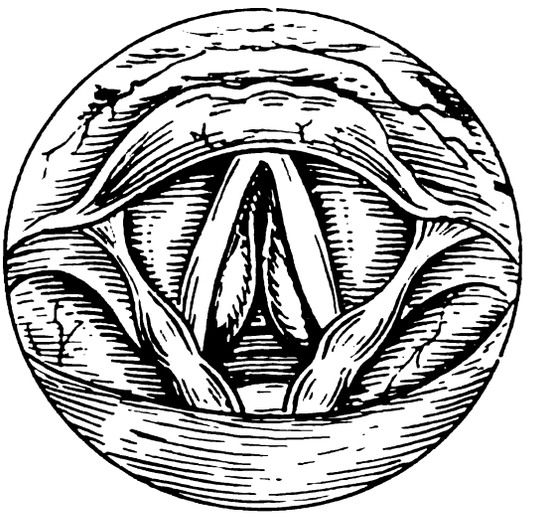


Fig. 99. False Croup (Subglottic Edema of Laryngeal Mucosa)

In children acute stenosis of the larynx is consequent upon false and true or diphtheritic croup (diphtheria). False croup usually follows the common cold or influenza with a sudden attack, most commonly at night when the child is asleep. The child awakens with a harsh cough and severe dyspnea. In severe obstruction to respiration, there is cyanosis, marked restlessness and sometimes convulsions. Attacks of asphyxia commonly last for a few minutes to half an hour, rarely for one or two hours, and may recur several times during the night. In cases of false croup, aphonia is absent because edema is confined to the subglottic area (Fig. 99). The quick aggravation of asphyxia is due to the narrowness of the lumen of the larynx and trachea in children.

In laryngeal diphtheria, known as true, or diphtheritic, croup, symptoms of asphyxia and dyspnea develop slowly and are accompanied by aphonia. The severe malaise and a number of additional symptoms indicative of intoxication serve to differentiate false croup from diphtheria of the larynx.

Laryngeal edema, particularly in children, always requires urgent medical aid. The child must be immediately seated and the room hung with wet bed-sheets to dampen the air. The feet should be put in a hot bath or mustard plasters applied to the calves. Sudorifics are also helpful. Timely precautions often forestall a second attack in false croup. Only in very rare cases are intubation and tracheotomy required for children with false croup, if revulsants and tranquillisers are ineffectual. The treatment of diphtheria of the larynx consists in an immediate injection of antidiphtheric serum. In case of stenotic respiratory obstruction, intubation or tracheotomy should be performed without waiting for the extreme phase of asphyxia.

Adults with edema of the larynx must also be given revulsants, hot foot baths, mustard plasters, etc. Inhalation may be prescribed to dilute the sticky sputum. An effective remedy is intramuscular injection of penicillin in 100,000 unit doses once in every four hours. If conservative treatment fails, as sometimes happens, tracheotomy will be required.

The stricture of the glottis may be associated with a number of causes unconnected with an acute inflammation in the larynx. These may refer to paralysis of both dilator muscles of the glottis, present in typhoids, syphilis or in an operation on the thyroid gland, or a brief glottic spasm which sets in as a result of the larynx being painted with various medications or of inhalation of irritant gases, etc.

In chronic stenosis occurring in tumours, tuberculosis and syphilis of the larynx, dyspnea is of slower onset, due to the adaptation of the body to oxygen insufficiency.

Treatment. The treatment of laryngeal stenosis has two aims. One is to give the patient immediate relief from asphyxia and the other is to remove the causes of stenosis. The earliest possible use of penicillin by intramuscular injection is indicated in all inflammatory processes. In serious cases of stenosis immediate aid may consist of intubation or tracheotomy, which must be done without delay to prevent heart failure due to oxygen deficiency and general weakening of the body as a result of continued resistance to stenosis.

MOTOR DISORDERS OF THE LARYNX

Motor disorders of the laryngeal muscles may be present with a marked increase in their function or, on the contrary, with decreased activity and full depression, i.e., paralysis.

Reflex hypersensitivity of the neuro-muscular system of the larynx is commonly met with in children, and is symptomatic of constitutional diseases, such as spasmophilia, rickets, whooping cough, etc. It is marked by spasmodic closure of the glottis with signs of cyanosis which may recur many times. A laryngospasm may be brought about by a reflex irritation of the larynx by a foreign body, the use of cautery, inhalation of a harmful and irritant gas, etc. In other cases, the laryngeal spasm may originate in the central nervous system, as, for instance, in hysteria, epilepsy, tabes dorsalis, tetanus, and other diseases. The aim of the treatment must be to cure the principal disease.

A weakening, or paralysis, of the laryngeal muscles may be associated with their lesions or disturbed nerve supply.

Distinction should be made between functional paralyses, which in most cases are caused by affections of the constrictor muscles of the glottis, and organic paralyses which are due to lesions of the laryngeal nerves, above all of the dilator muscles of the glottis.

Disturbances of the laryngeal motor function may originate both in the central and the peripheral nervous systems. The cause of *central paralysis* may be syringomyelia, tabes, hysteria, as well as gummas, tumours, hemorrhages in the cerebral cortex, bridge of Varolius, medulla oblongata, and sometimes in other parts of the brain stem. Sometimes, inferior laryngeal nerve paralysis of central origin is accompanied by simultaneous lesions of other neighbouring cranio-cerebral nerves, viz., the 9th, 10th, 11th and 12th.

If a lesion of the central nervous system is suspected, the appropriate laryngoscopic picture will provide additional data for confirming the diagnosis of the main disease.

Peripheral paralysis follows an injury to the recurrent laryngeal nerve which on its relatively long path may be compressed by mediastinal tumours, aortic aneurysms, goitre and carcinoma of the esophagus, or it may result from

affection of the nerve itself, such as alcoholic and syphilitic neurites in tabes, and neuritis of rheumatic origin. Lesions of the inferior laryngeal nerve are frequently caused by excision of the goitre. Laryngeal examination reveals that the vocal cord on the paralysed side, instead of being abducted, lies half-way between the position during respiration and during phonation, that is, in the intermediate position, otherwise known as the cadaveric position (Fig. 100, a, b).

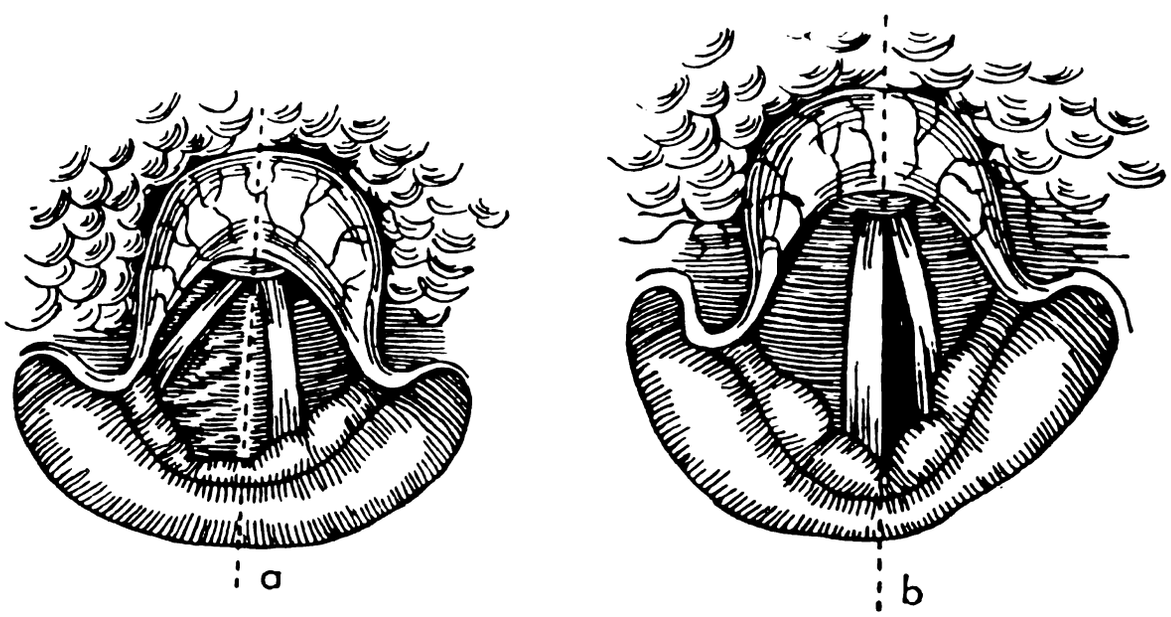


Fig. 100. Paralysis of Left Side of Larynx
(a) in quiet respiration; (b) in phonation

The clinical symptoms of unilateral paralysis of the recurrent nerve are slight. The affection of the vocal chords is relatively mild with slight hoarseness, quick vocal fatigue and free respiration. Bilateral paralysis, however, endangers the patient's life and often requires tracheotomy, since both cords lie so close to the median line as to narrow the glottis to the point of asphyxia.

Apart from neuropathic or organic paralyses of the larynx there are frequent *myopathic, functional paralyses* caused by all kinds of inflammations in the larynx or vocal abuse by public speakers, singers, teachers, etc. The lesion more often affects the vocal muscles.

Paresis of both vocal cords prevents their full approximation in phonation, and the glottis in such cases is a long

and oval chink pointed at both ends (Fig. 101). The voice becomes hoarse, in some cases there may be complete aphonia.

It should be noted that myopathic and neuropathic paralyses are clinically very much alike and offer completely identical signs in laryngoscopy. It should also be borne in mind that paralysis of the inferior nerve is a symptom of constitutional, and perhaps very serious disturbance.

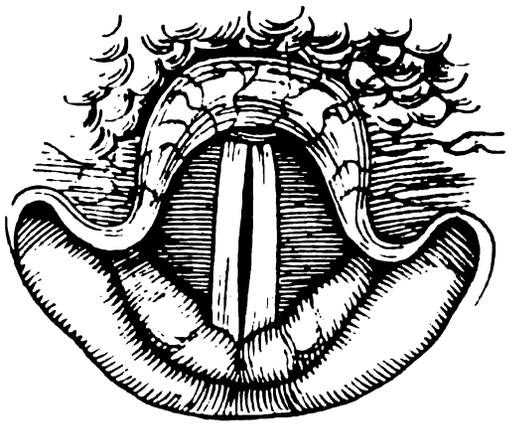


Fig. 101.
Bilateral Paresis
of Vocal Muscles

Treatment. The primary measure is to remove the causes of the disease. Prolonged vocal rest, treatment of chronic inflammation and the wide use of electrotherapy with galvanic and faradic currents may be recommended to hasten cure. At the onset of the disease, these measures are usually effective. The chances of recovery from neuropathic paralysis of the laryngeal muscles are strictly contingent on the outcome of the basic disease.

TUBERCULOSIS OF THE UPPER RESPIRATORY TRACT

Tuberculosis of the Nose
(*Tuberculosis nasi*)

Tuberculosis of the nose is most commonly a secondary infection due to a primary focus in the lungs. Primary tuberculosis of the nose occurs seldom.

This condition presents as infiltration or ulceration in the nasal cavity. The infiltration may be of variable size, and is sometimes the shape of a tumour (tuberculoma). The breakdown of tubercles and infiltration on the mucosa leads to ulceration. Tuberculous ulcers are most frequently confined to the cartilaginous part of the nasal septum.

At the onset of the disease, subjective symptoms are almost completely lacking. The increase of infiltration with the progress of the disease brings about a more or less severe nasal obstruction. The breakdown of the infiltration and

ulceration are followed by a purulent and sometimes blood-stained nasal discharge, which may turn into crusts. The discharge is commonly odourless. The diagnosis of nasal tuberculosis often requires confirmation by biopsy in order to differentiate the incipient ulceration from a malignant tumour.

Tuberculosis of the Pharynx
(*Tuberculosis pharyngis*)

Tuberculosis of the pharynx is a secondary infection brought with the sputum in cavernous pulmonary tuberculosis or carried by the blood and lymph flow. The stage of tuberculous infiltration of the pharynx often causes no marked subjective symptoms, and, therefore, the very first examination of the patient may reveal tuberculous ulcers. These are confined to the soft palate, faucial tonsils and posterior pharyngeal wall, and less frequently they invade the tonsils. Tuberculous ulcers are mostly shallow with "mouse-nibbled", or crenate, undermined margins of a pale pink colour.

These ulcers coalesce and rapidly invade extensive areas. There is pain on swallowing. Before the advent of streptomycin and other new antibiotics as well as antituberculous drugs, such as PAS, phthivasid, etc., the treatment of the disease was prolonged and often unsuccessful.

Tuberculosis of the Larynx
(*Tuberculosis laryngis*)

Tuberculosis of the larynx is secondary to pulmonary tuberculosis. In chronic fibro-cavernous pulmonary tuberculosis the infection occurs through the accumulation of tuberculous sputum in the larynx, as well as in fissures and places of desquamation of the epithelium. In patients with other forms of pulmonary tuberculosis the infection is usually carried by the blood and lymph flow. A vocal cord or the interarytenoid area is by far the most common initial site of invasion, where the mucosa is swollen and congested. The process goes deeper while the infiltration increases and breaks down to form ulcers growing in area

and depth. Concurrently with infiltration the cartilage and perichondrium are involved in the necro-inflammatory process in other places with an open danger of laryngeal edema and associated respiratory obstruction requiring tracheotomy.

An infection entering by the hematogenous route will cause eruption of tubercles on the laryngeal mucosa, often simultaneously occurring in the fauces and laryngeal aperture. The hematogenous form has a faster course, smaller incidence and is frequently accompanied by dysphagia.

Symptoms. Patients with laryngeal tuberculosis in the incipient stage are commonly susceptible to repeated laryngeal catarrhs, which come on readily for most insignificant reasons and persist longer than usual. These laryngeal catarrhs are most likely aggravations of the tuberculous process. With the appearance of infiltration and ulcers the voice becomes hoarse, the cough goes from bad to worse, there is pain on swallowing, which in ulceration of the epiglottis and arytenoid cartilages may be so excruciating as to make it impossible for the patient to eat.

Treatment. In the past few years there has been considerable progress in the treatment of tuberculosis, particularly that of the upper respiratory tract. New antibacterial drugs have given fresh hope of clinical recovery from many forms of tuberculosis, including those formerly considered almost incurable, such as the ulcerative form of laryngeal tuberculosis. Streptomycin, PAS and other effective antituberculous drugs are widely used in the treatment of acute and aggravated chronic tuberculosis of the upper respiratory tract. The best remedy for the hematogenous forms of laryngeal tuberculosis is streptomycin which gives early relief from dysphagia and soon leads to recovery. Sanatorial and climatic treatment is also an integral part of combination therapy for tuberculosis.

The first requirement in laryngeal tuberculosis is to ease the larynx by vocal rest, unforced speech, to observe a bland diet, and to abstain from tobacco and alcohol.

Infusions of the larynx with 1-3% menthol oil in 1 or 2 ml doses every other day or cautery of laryngeal ulcers with trichloracetic acid or galvanic current which were for-

merly used on a wide scale have to this day in some cases retained a certain significance.

With the onset of pain on swallowing anesthetics must be applied by insufflation, spraying or inhalation before every meal.

Here are the appropriate formulae:

Rp. Orthoformii 1.0
(seu Anaesthesini)
Acidi borici pulverati subtilissimi 2.0
MDS. For insufflation into the larynx

Rp. Cocaini hydrochlorici
Morphini hydrochlorici aa 0.2
Aq. destill.
Aq. Menthae aa 100.0
MDS. For a spray two or three minutes before the taking of food

Rp. Mentholi crystallisati 0.1
Anaesthesini 3.0
Spiritus vini rectificati
Acidi tannici aa 10.0
MDS. For lubricating the larynx

Prophylaxis and *nursing care.* The strict observance of sanitary and hygienic rules is an indispensable measure for efficient care of the patient. The normal air temperature of 18° to 19° C must be maintained in the room, which should be frequently ventilated all the year round, and the patient must have regular meals and take medicines at the appropriate times and in correct dosages. The importance of resting the larynx should be explained to the patient. The patient must be instructed to use disinfectant-filled spitoons for fear of the infection being communicated to other people.

Lupus of the Nose and Pharynx

(*Lupus nasi et pharyngis*)

A special form of tuberculosis in the nose and pharynx, lupus, develops as a complication of tuberculous lupus of the skin or tuberculosis of the lymph nodes and bones. This disease commonly produces inactive tuberculous foci in the lungs.

Although lupus of the nasal cavity is a comparatively

rare disease, it is still more frequent than lupus of the pharynx and larynx. In the nose it most commonly affects the nasal septum and wings. The characteristic signs of lupus are nodular infiltration and scarring with a simultaneous spread of the lesions to the facial skin.

Lupus of the pharynx occurs simultaneously with a similar condition on the face and in the nose. At first the pharyngeal mucosa is the site of flat, pale, and fine-grained infiltration which later turns into superficial ulceration. These ulcers give only slight pain or no pain at all and, unlike tuberculous ulcers, they tend to cicatrise. Lupus runs a chronic course and has little influence on the patient's general condition. Recommended treatment for lupus of the nose, pharynx and larynx is with vitamin D_2 or phthivasid, which has a tremendous curative effect on this disease.

The Soviet Union has a wide network of clinico-prophylactic centres for tuberculosis control known as "antituberculous dispensaries", where patients are hospitalised or placed under constant medical control. The obligatory prophylactic examination of the larynx of all patients with pulmonary tuberculosis by a staff laryngologist at any "dispensary" makes possible an early detection of laryngeal tuberculosis and the application of effective therapy.

The all-embracing system of compulsory "dispensary" treatment for all patients with tuberculosis and the use of new effective antibacterial and antituberculous remedies have drastically lowered the sickness and mortality rates of tuberculosis, especially that of the upper respiratory tract.

SYPHILIS OF THE UPPER RESPIRATORY TRACT

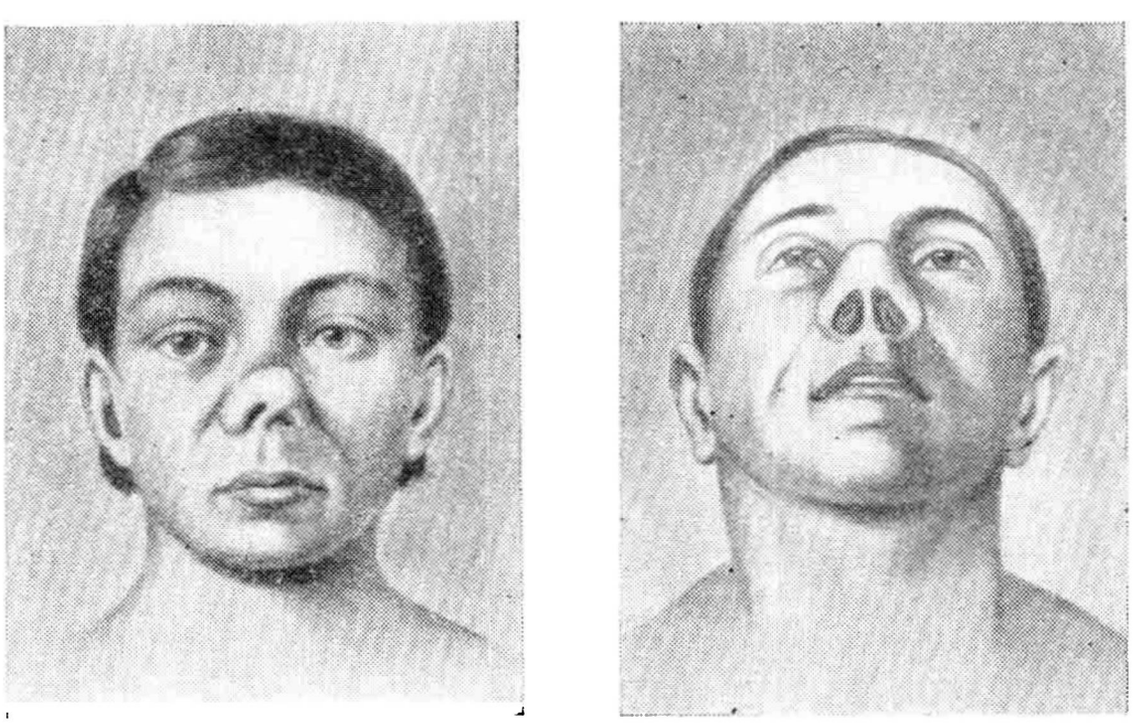
Syphilis of the Nose
(*Syphilis nasi*)

Syphilis of the nose presents as primary sclerosis or as lesions of the secondary and tertiary stages. The tertial stage of nasal syphilis is the most common stage seen in this disease, and, therefore, is a matter of special importance for the medical practitioner. Pathoanatomical examination

reveals limited or diffuse syphilitic infiltrations, the so-called gummas, which subsequently disintegrate. Destruction of syphilitic infiltrate formed in the mucous membrane gives rise to a deep ulcer with sharply outlined edges and a sebum-lined bottom. This ulcer may later invade the underlying bone and cartilage. If the primary site of the gumma is in the bone or periosteum, the trophic disturbance in the bone will bring about its necrosis with sequestration, accompanied by a revolting fetor.

Tertial syphilis may affect all tissues of the nose but the favourite site is the bony part of the nasal septum which is liable to perforation by gummatous breakdown. The destruction may also involve the floor of the nasal cavity and form a hole between the latter and the oral cavity. Intracranial complications may result from necrosis of the cribriform plate of the ethmoid bone.

In neglected cases, there are characteristic nasal deformities caused by destruction of the nasal septum and nasal bones with a subsequent sinking of the nasal bridge and cicatricial contraction of connective tissue. This deformity is known as the saddle-nose or sunken nose (Fig. 102, a).



a b

Fig. 102.

(a) syphilis of nose; (b) scleroma of nose

Diagnosis. In the absence of other signs of syphilis on the body, the early tertial stage may be somewhat difficult to identify.

Treatment. This should above all be general and specific. A delay in specific treatment will thwart all efforts to prevent the extremely severe destruction of the nose.

External nasal deformities, like the saddle-nose, are best corrected by injections of plastic paraffin or by intranasal transplantation of bony or cartilaginous plates taken from the tibia or costal cartilage, as well as by the grafting of cadaveric cartilage.

Syphilis of the Pharynx
(*Syphilis pharyngis*)

Syphilis of the pharynx is encountered in all of its three forms.

The *primary lesion* is found on the tonsils or the posterior pharyngeal wall. Pain is usually absent. Specific lymphadenitis of the cervical and occipital glands which become dense and swollen, even if painless, develops after some time and helps to establish the correct diagnosis.

The *secondary symptoms* of syphilis in the pharynx are usually those of syphilitic angina characterised by a low-grade fever, a very mild pain, and diffuse copper-hued congestion invading the faucial pillars, as well as the soft and hard palates. The disease has a subacute course and continues for weeks.

In other cases, broad condylomas or papules may be discovered at this period. Papular rashes appearing as greyish-white and slightly prominent patches of round or oval shape ringed with a red border spring up on the markedly reddened faucial pillars, the palate or tonsils. The regional lymph glands are always swollen.

Tertiary syphilis occurs either as a limited gummatous tumour or as a diffuse infiltration with congestion along its circumference. Some time after their breakdown the gummatous formations turn into a deep gummatous ulcer with even edges and a sebum-lined floor covered with necrotic tissue.

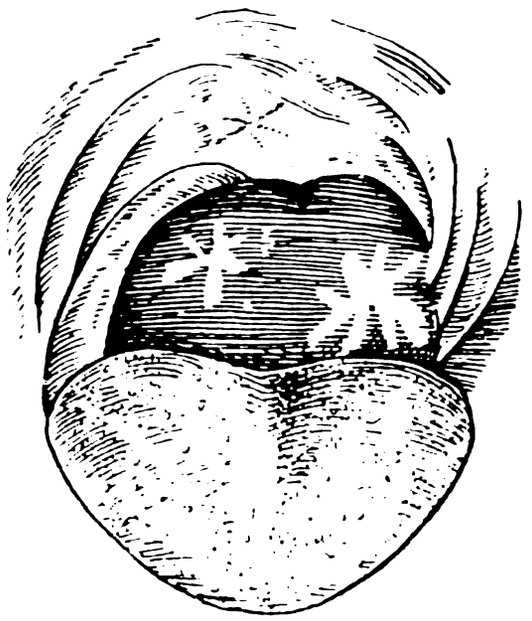


Fig. 103. Syphilitic Star-Shaped Scars of Soft Palate and Posterior Pharyngeal Wall

Failure to give timely treatment will cause a further destruction of the soft and bony tissues of the pharynx, perforation of the soft and hard palates, breakdown of the faucial pillars, etc.

Treatment is usually followed by healing with formation of characteristically star-shaped and compact scars, while accretions and perforations remain (Fig. 103).

Treatment. This must be specific. Locally, mildly antiseptic gargles are used, such as hydrogen peroxide, potassium chlorate, etc.

Syphilis of the Larynx
(*Syphilis laryngis*)

Syphilitic symptoms are found in the larynx when the disease is in the secondary or tertiary stage. In secondary syphilis the larynx becomes reddened and covered with papular eruptions that appear in characteristically raised white patches which after their breakdown remain as separate but interconnected superficial ulcers. More commonly, however, the secondary symptoms occur in the larynx as a syphilitic erythema which may be differentiated from an acute laryngeal inflammation only by the presence of other signs of secondary syphilis on the body. The latter may be evident from three weeks to three years after infection.

The gummatous or tertiary stage is characterised by gummatous infiltrations and tumours, whose breakdown gives rise to secondary perichondritis of the laryngeal cartilages and stubborn cicatricial changes. The primary lesion of the cartilage causes still greater destruction. The favourite site of affection is the epiglottis, which sometimes utterly disappears during the process. The laryngoscopic pic-

ture of the larynx in tertiary syphilis is extremely varied. The breakdown of individual gummatous nodes produces a deep ulcer with peculiar sharply outlined edges, a sebum-lined floor and a surrounding band of inflamed, infiltrated tissue. This typical picture is often changed by the added symptoms of secondary infection and makes diagnosis difficult. Ulceration and destruction of the larynx give little pain until being joined by secondary infection which causes a phlegmonous inflammation or perichondritis of the laryngeal cartilages. The scarring leads to persistent laryngeal stenosis, for which tracheotomy may be needed.

The diagnosis rests on the history, local symptoms, general examination and Wassermann's test. Biopsy is used in obscure cases where a differential diagnosis has to be made between syphilis, tuberculosis and laryngeal cancer. It should be borne in mind that the use of various cauterising agents, particularly silver nitrate solutions, before the condition has been identified will distort the symptomatic picture and may lead to diagnostic errors. As long as the diagnosis has not been made only bland remedies may be used, such as gargles and douches of physiological solutions, oily solutions for the throat, etc.

Treatment. This is specific and in the past few years has been largely based on penicillin. If specific treatment fails to relieve stenosis the patient must be watched closely, so that an urgent need for tracheotomy may not be overlooked.

Caution must be displayed in examining syphilitic patients, particularly in the second stage, where the disease is the most contagious. Quite a few cases of infection through aural catheters, probes, spatulas, and the like have been described in literature. Separate instruments must be reserved for such patients and thoroughly sterilised by boiling after each examination. Following various procedures employed in lubricating the syphilitic patient's pharynx and larynx, the cotton must necessarily be picked off the probe with pincers and either dropped in a sublimate-filled spittoon or burned immediately.

The U.S.S.R. has a ramified network of dermato-venereological dispensaries dealing with the detection, treatment

and further observation of all syphilis cases. In the U.S.S.R. the incidence of this disease is decreasing owing to the work of specialised medical institutions which are tackling the problems of therapy together with those of prophylaxis.

SCLEROMA

Scleroma is a chronic infectious disease of the upper respiratory tract in the form of quickly cicatrising infiltrations on the mucous membrane. It is caused by Volkovich-Frisch's bacillus. Russian scientists have been to the fore in devoting extensive study to the problems of scleroma (transmission of the disease, problems of diagnosis and therapy).

The disease is commonly preceded by a chronic inflammation of the mucosa, which serves as a background for infiltrations in the nose, pharynx, larynx and sometimes the trachea and bronchi. The infiltrations appearing as flat or knobby prominences of a pale red colour are usually confined to organically narrow structures, such as the nasal vestibule (see Fig. 102, b), choanae, nasopharynx, and subglottic space. These infiltrations later transform into scar tissue without any breakdown but simply in consequence of proliferation of connective tissue cells, and form characteristic adhesions and markedly narrow spaces. At this stage, some patients emit a very peculiar and weak odour from the nose and mouth.

The patient's basic complaint is one of a respiratory disorder due to a concentric cicatricial contraction of the affected part of the upper respiratory tract, as well as of aphonia in cases of infiltrations in the larynx and dryness of all mucous membranes of the upper respiratory tract.

Diagnosis. This is made by examination of the upper respiratory tract and bronchoscopy and rests on the clinical symptoms and absence of ulcerations in prolonged observation. In case of a differential diagnosis, syphilis must be excluded. Sometimes, biopsy and Bordet-Gengou's serological reaction with the scleromal antigen have to be used.

The disease may last for years and become worse.

Treatment. The antibiotic treatment of scleroma, with streptomycin and terramycin in particular, practised over the past few years has met with considerable success. Streptomycin is given by intramuscular injection in 500,000 unit doses twice a day, the overall dose being up to between 10 and 20 to 40 million units according to the gravity of the condition. Patients in the initial stage of scleroma with small infiltrations and of comparatively short duration are now usually certain to recover. Many cases of decrease or disappearance of infiltrations with amelioration in the general condition have also been noted in patients with well-advanced scar formation. It has been possible to remove the cannula from a number of tracheotomised patients.

Streptomycin therapy has been considerably assisted by X-ray treatment and bouginage of the larynx and trachea when found necessary.

Prophylaxis. Scleroma is a heritage of the past. The growth in public welfare and cultural standards, as well as the planned measures of health organisations in areas where the incidence of scleroma is high comprising the detection of scleroma cases, deep study of its clinical picture and various predisposing causes, and large-scale in- and out-patient services have reduced the incidence of this disease in the U.S.S.R. and will undoubtedly result in its full eradication in the years to come.

DISEASES OF THE TRACHEA

ANATOMY OF THE TRACHEA

The trachea is the initial portion of the lower respiratory tract, and is a continuation of the larynx. It is an 11 to 13 cm long cylindrical tube descending from the larynx to the point of bifurcation and consisting of 16 to 20 tracheal rings made of hyaline cartilage and kept together with dense fibrous tissue. Posteriorly, where the tracheal rings are not fully closed, the tracheal wall is formed of connective tissue and smooth muscles. This part of the trachea adjoins the esophagus all along its length.

At rest, the trachea in the adult lies at the level of the seventh cervical or first thoracic to the fifth thoracic vertebra. The connective tissue around the trachea is loose and allows large displacements in the movement of the larynx and trachea. In its upper portion, the trachea lies closer to the surface of the neck; it then extends downwards and backwards, and is covered with a mass of well-developed fat and connective tissue, the anterior cervical muscles, superficial fascia and skin.

TRACHEOBRONCHOSCOPY

Tracheobronchoscopy is a modification of direct laryngoscopy. For examination of the trachea and bronchi, metal tubes are inserted into the respiratory passages as deep as the second and third bronchial divisions. The bronchoscopic set (Fig. 104) is made up of the following parts: (1) a handle with a lighting equipment, (2) bronchoscopic tubes, (3) auxiliary and surgical instruments, including threaded probes for wiping off mucus and blood, a mucus aspirator and a long forceps with different extension pieces (Fig. 105) for

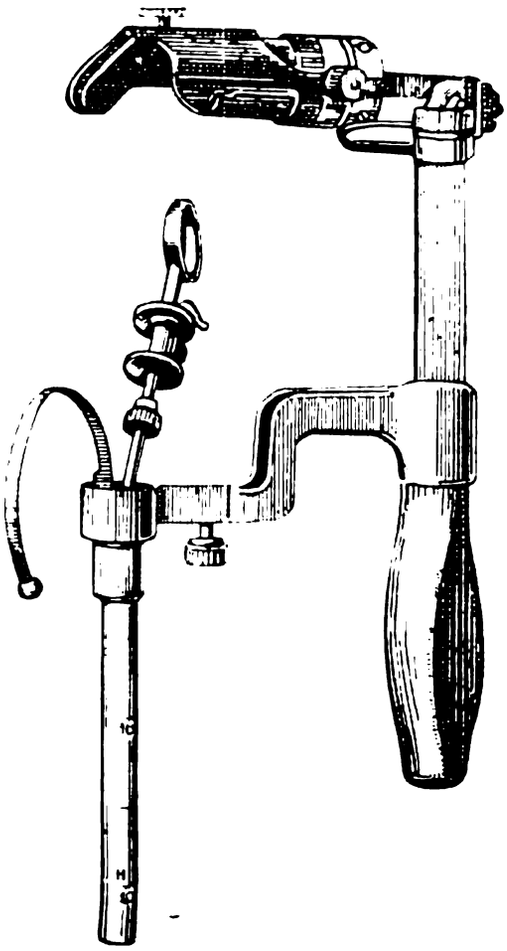


Fig. 104. Bronchoscope with Forceps Inserted into Bronchoscopic Tube

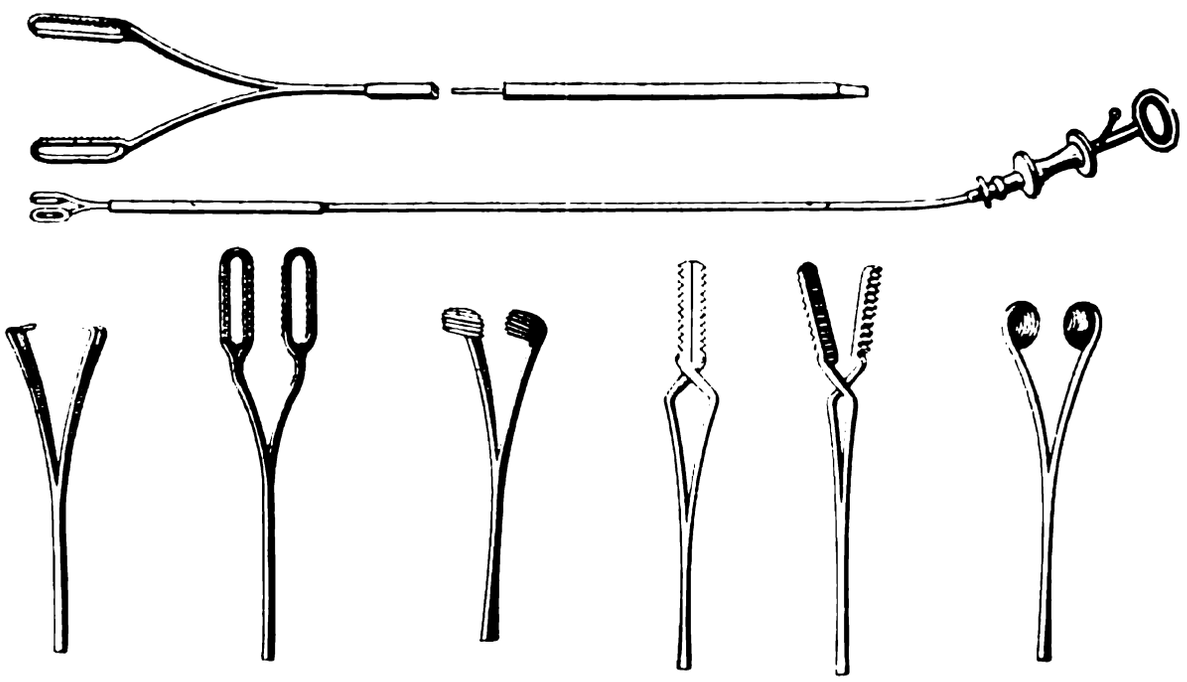


Fig. 105. Bronchoscopic Forceps with Different Extension Pieces for Extraction of Foreign Bodies

the extraction of foreign bodies and other operations on the bronchi.

Bronchoscopic tubes may be inserted into the lower air passages for their lighting and examination either by superior bronchoscopy, that is through the natural openings, such as the mouth, pharynx and larynx, or by inferior bronchoscopy where the tubes are inserted through a tracheotomy hole, if present. The latter procedure is more commonly used for children under five or six years of age after preliminary tracheotomy. Bronchoscopy is now widely used in the diagnosis of diseases of the trachea and bronchi, and in treatment of a number of pulmonary diseases, such as lung abscesses, bronchiectasis, etc.

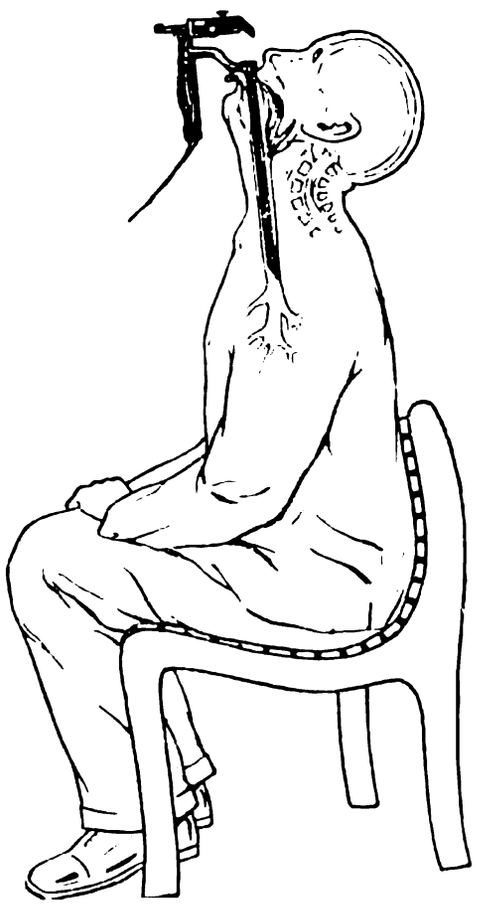


Fig. 106. Bronchoscopy in Sitting Posture

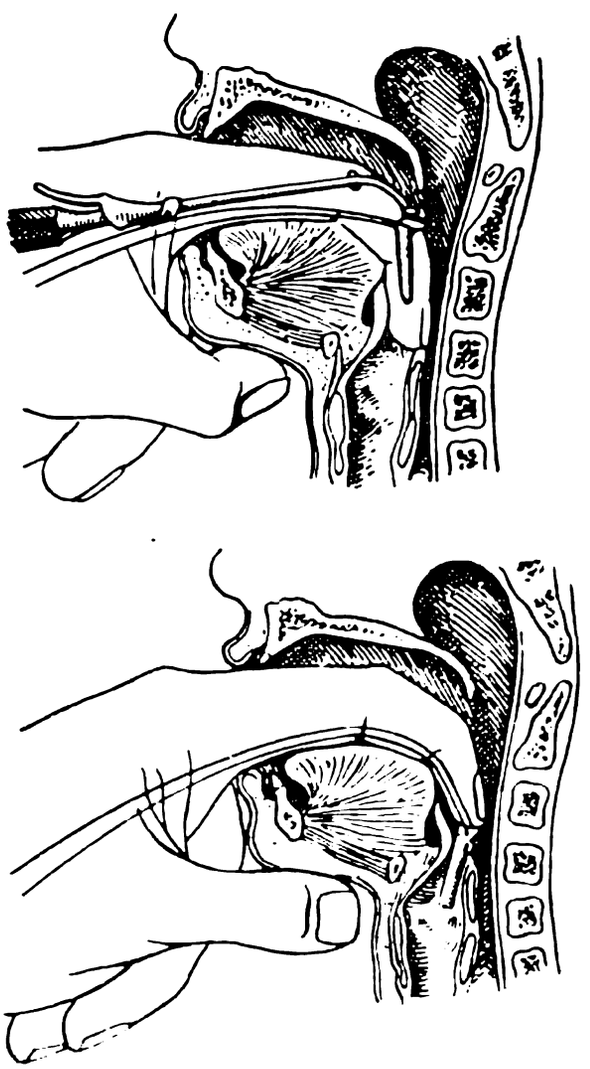


Fig. 107. Intubation of Larynx

INTUBATION

Intubation (Fig. 107) is most commonly used for children with transitory stenosis, for instance, in diphtheria, false croup, edematous reaction of the larynx to insect bites, cauterisation, etc. In intubation the child is seated and kept in position in the same manner as in adenotomy. A

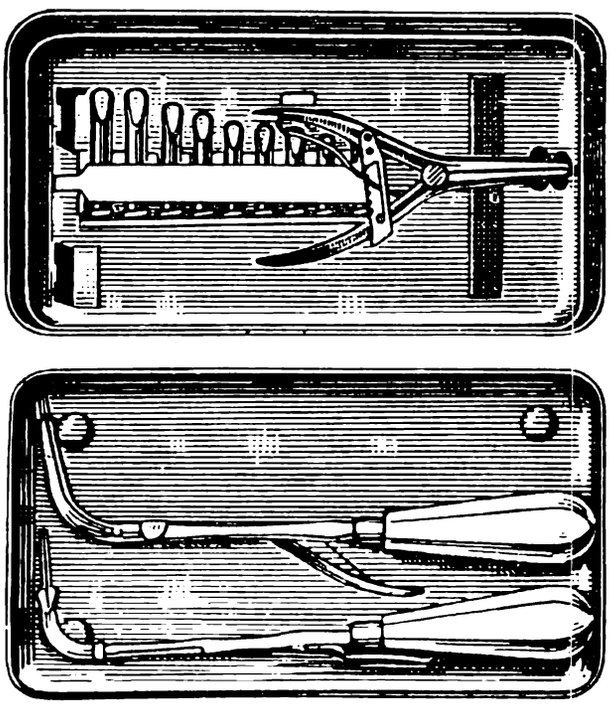


Fig. 108. Laryngeal Intubation Set Consisting of Gag, Intubator, Extubator, and Selection of Intubation Tubes of Different Diameter

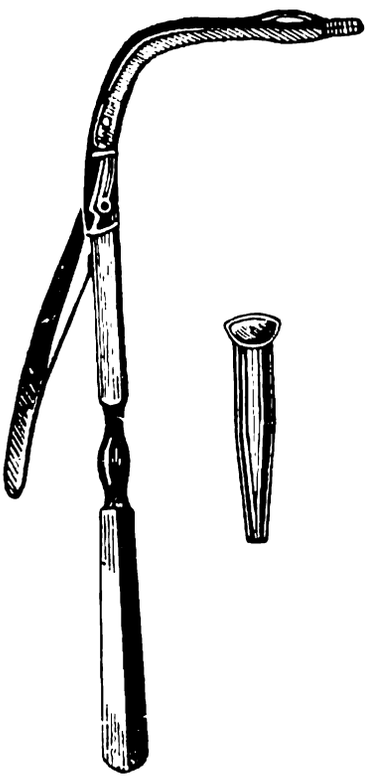


Fig. 109. Intubation Tube and Intubator

hollow metal tube is passed with a special instrument called the intubator (Figs. 108 and 109) into the laryngeal orifice along the forefinger of the left hand inserted into the larynx. If the tube has slipped into the larynx, there will be cough and free respiration through the tube with a characteristic whistling sound. For easy removal of the tube it is tied in advance to a silken thread whose free end is glued to a cheek with an adhesive plaster. Intubation is inconvenient in that the tube has to be removed in 24 to 36 hours to avoid sores in the larynx. Another disadvantage is the need for constant vigil by a doctor, since the tube may slip out easily during coughing or vomiting, while

a restless child may just as easily pull it out by the thread. Nevertheless, it has its own advantages over tracheotomy in that it can be carried out more quickly and requires fewer assistants.

TRACHEOTOMY

Tracheotomy is an operation performed in cases of severe stenosis of the larynx or upper part of the trachea caused by croup, laryngeal edema, foreign bodies, tumours, etc., which endanger the patient's life. All physicians regardless of their specialities must be able to perform tracheotomy.

An early and common symptom of laryngeal stenosis is inspiratory dyspnea with characteristic hindrance to the free passage of air into the lungs.

Stenosis has three stages. The first, or the so-called compensation stage, is marked by an intensive activity of the respiratory muscles whereby the patient tries to compensate for air deficiency. At this stage breathing is deep and sparse, and the physiological pause between inspiration and expiration disappears. There is stenotic breathing which is easily heard at a fairly long distance, sometimes from a neighbouring room. Then, auxiliary muscles are brought into play, and the tissue filling the intercostal spaces becomes indrawn. After a time these reserves are no longer sufficient, and this marks the onset of the second stage, that of decompensation. The skin, above all the facial skin, becomes cyanosed, and later becomes markedly pale. The patient grows restless and opens the mouth involuntarily in an effort to make up the air deficit; his breathing progressively quickens and he becomes covered by a cold perspiration.

Failure to give the patient appropriate surgical aid at this stage of stenosis may be followed by the onset of the third stage, asphyxia; respiration becomes slow and shallow, cardiac activity decreases and the patient becomes apathetic and finally loses consciousness.

Tracheotomy is indicated at the second stage of stenosis. When the patient is still in the compensation stage, a number of therapeutic measures should be attempted to relieve the acute local inflammation and remove the symptoms of stenosis by the application of revulsant remedies, like a hot bath for the feet or a mustard plaster, as well as by the intra-

venous injection of 40% glucose solution, intramuscular injection of large doses of penicillin, etc. Meanwhile, the patient must be kept under close medical observation in order that the moment of transition to the second stage is not overlooked. Very often, however, the operation has to be performed at the third stage. Experience has proved that even at this stage prompt tracheotomy can save a life.

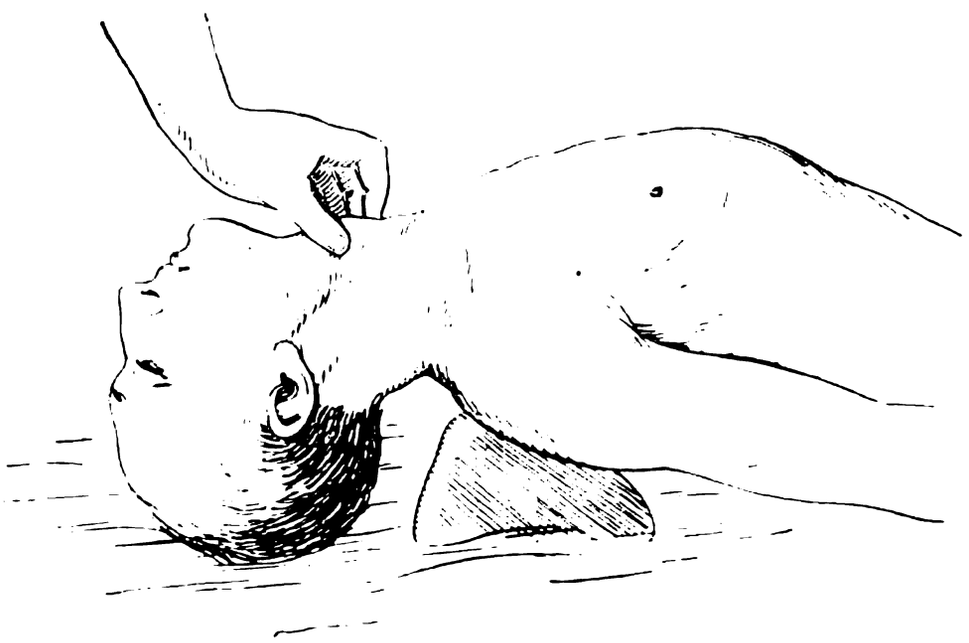


Fig. 110. Position of Patient in Tracheotomy

Tracheotomy may be superior or inferior depending on whether the trachea is opened above or below the isthmus of the thyroid gland. The patient is placed on the operating table with his shoulders propped high on a round bolster and his head tilted far back (Fig. 110). The skin and superficial cervical fascia are incised strictly in the mid-line of the neck, and the incision is carried from the lower edge of the thyroid cartilage some 6 cm downwards. The front surface of the cricoid cartilage is then exposed with blunt instruments strictly in the mid-line, a transverse incision made in the capsule of the thyroid isthmus lying below, and the isthmus pushed down to expose the first tracheal rings. Following the arrest of bleeding, two or three tracheal rings are cut with a sharp scalpel for insertion of the tracheotomy tube. This consists of two connected metal tubes which slide one within another. The insertion of the tube is followed

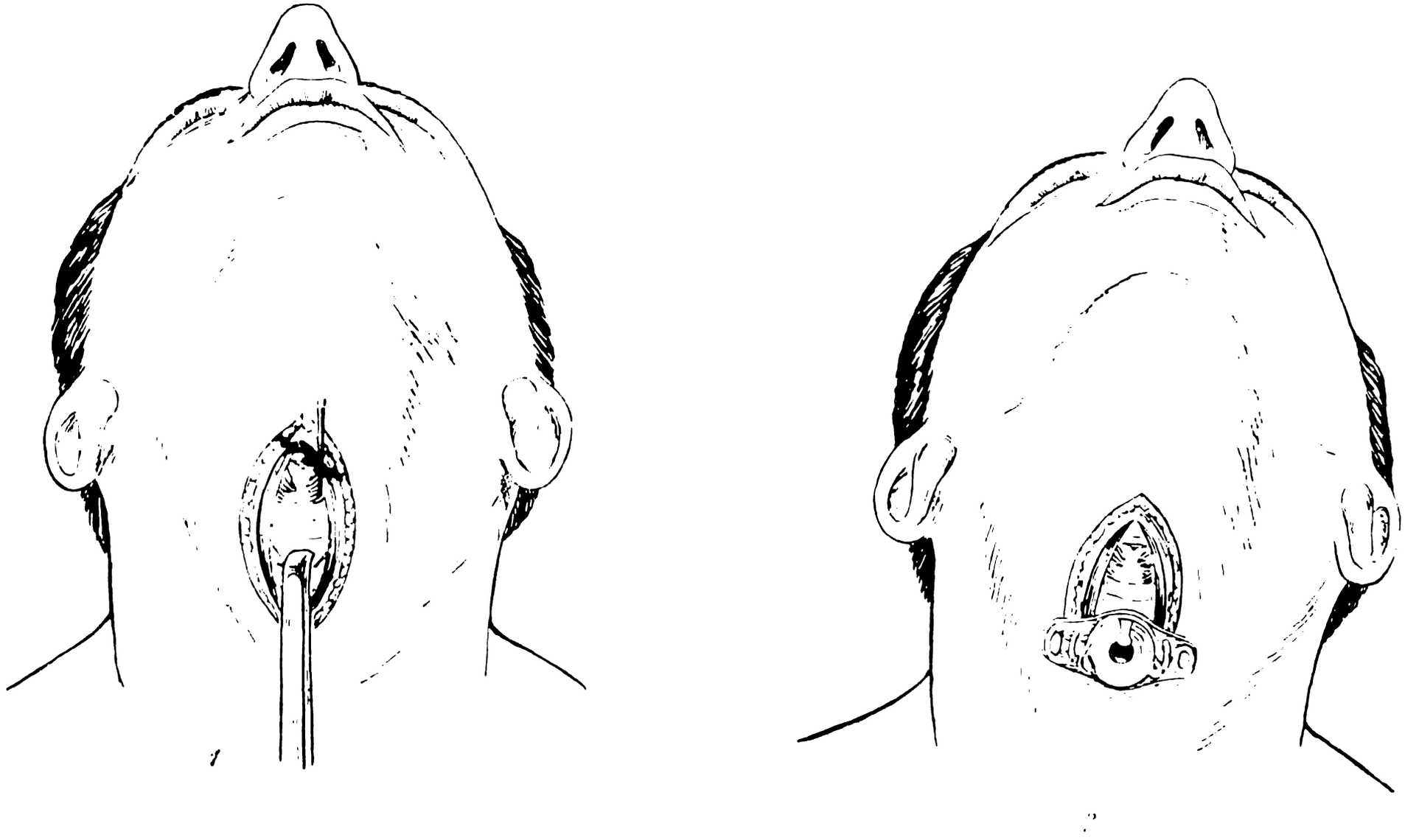


Fig. 111. Tracheotomy

(1) exposure of tracheal cartilages; (2) opened trachea with inserted tracheotomy tube

by a vigorous expectoration of sputum and then by quiet respiration. The tube is fastened with a bandage applied to the neck, while the incision is sutured with one or two stitches above and below the tube (Fig. 111, 1, 2 and Fig. 112).

The operation is commonly performed under local anesthesia but in the event of asphyxia where time is a factor of overriding importance no anesthesia is applied. In such patients general sensitivity is markedly diminished owing to carbon dioxide intoxication. During the first three or four days no attempt must be made to remove the entire tube from its position in view of the inflammatory edema of the tracheotomy passage and its rapid constriction. In such cases the tube may lodge between the trachea and the surrounding muscles or esophagus.

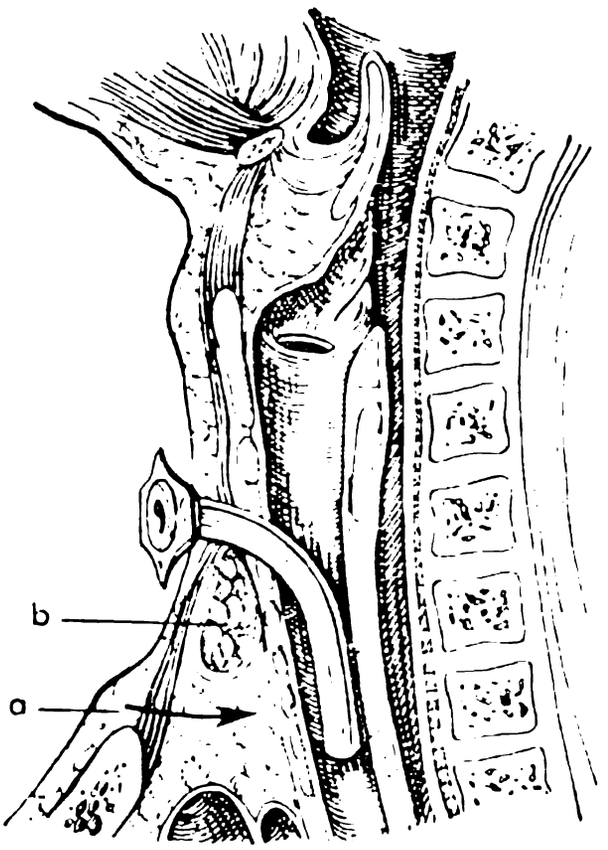


Fig. 112. Superior Tracheotomy
(a) arrow shows point of opening of trachea in inferior tracheotomy; (b) isthmus of thyroid

Organisation of emergency aid and *aftercare in tracheotomy.* For timely and adequate medical aid to stenotic patients every surgical office and centre for infectious diseases must always have a complete sterile set of tracheotomy instruments kept in the appropriate antiseptic conditions. This set must include blunt and sharp hooks, a few arterial clamps, two surgical and two anatomical pincers, tracheotomy tubes of various sizes, a retractor for the tracheotomy wound, a needle holder with a needle and sterile thread, a grooved director and several scalpels (Fig. 113).

Every operating room must have a complete set of this kind, which must always be ready for use and bear a corresponding identifying tag. In addition, 0.5% novocaine solution and a sterile syringe with a set of needles must be available in case of local anesthesia being applied should there

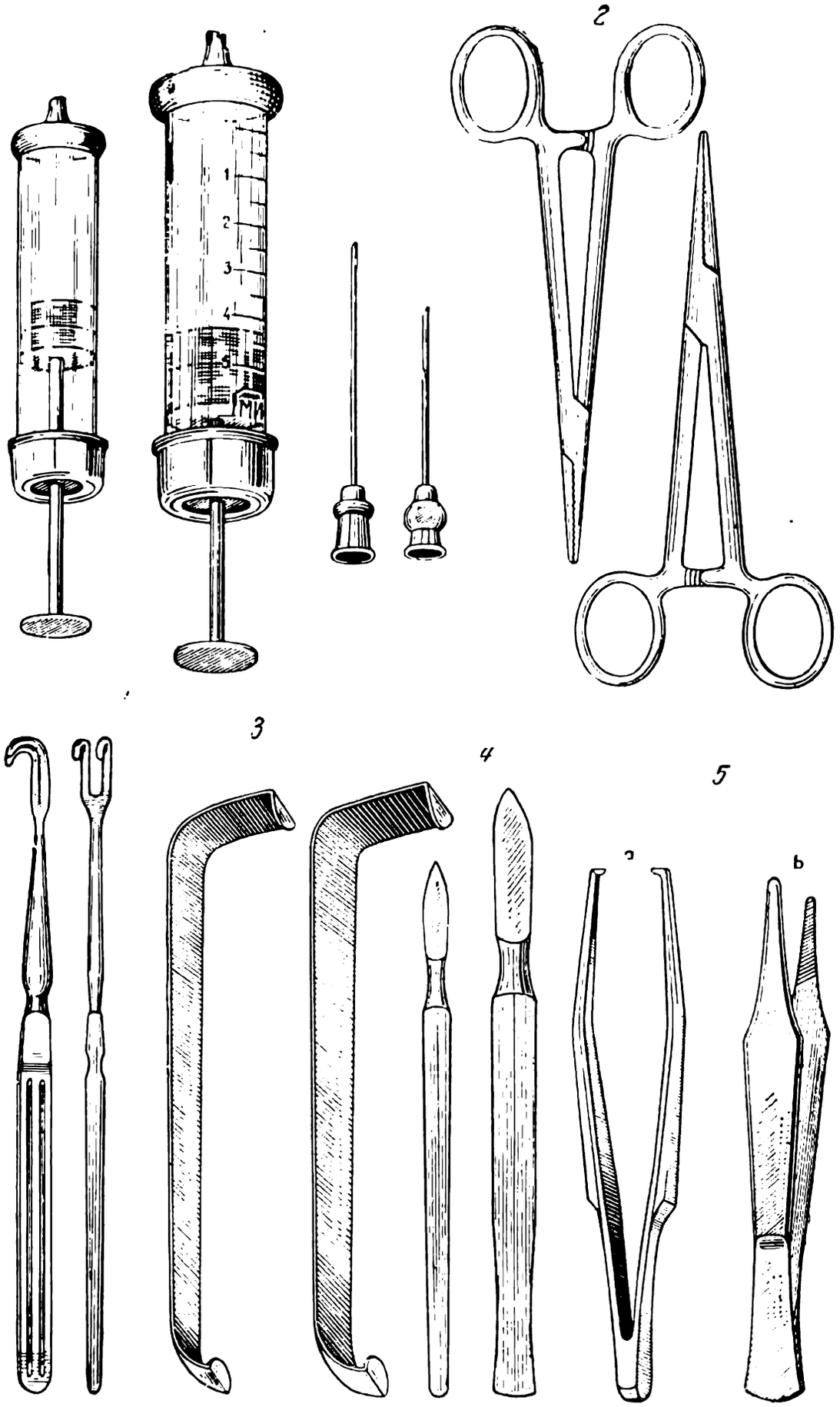
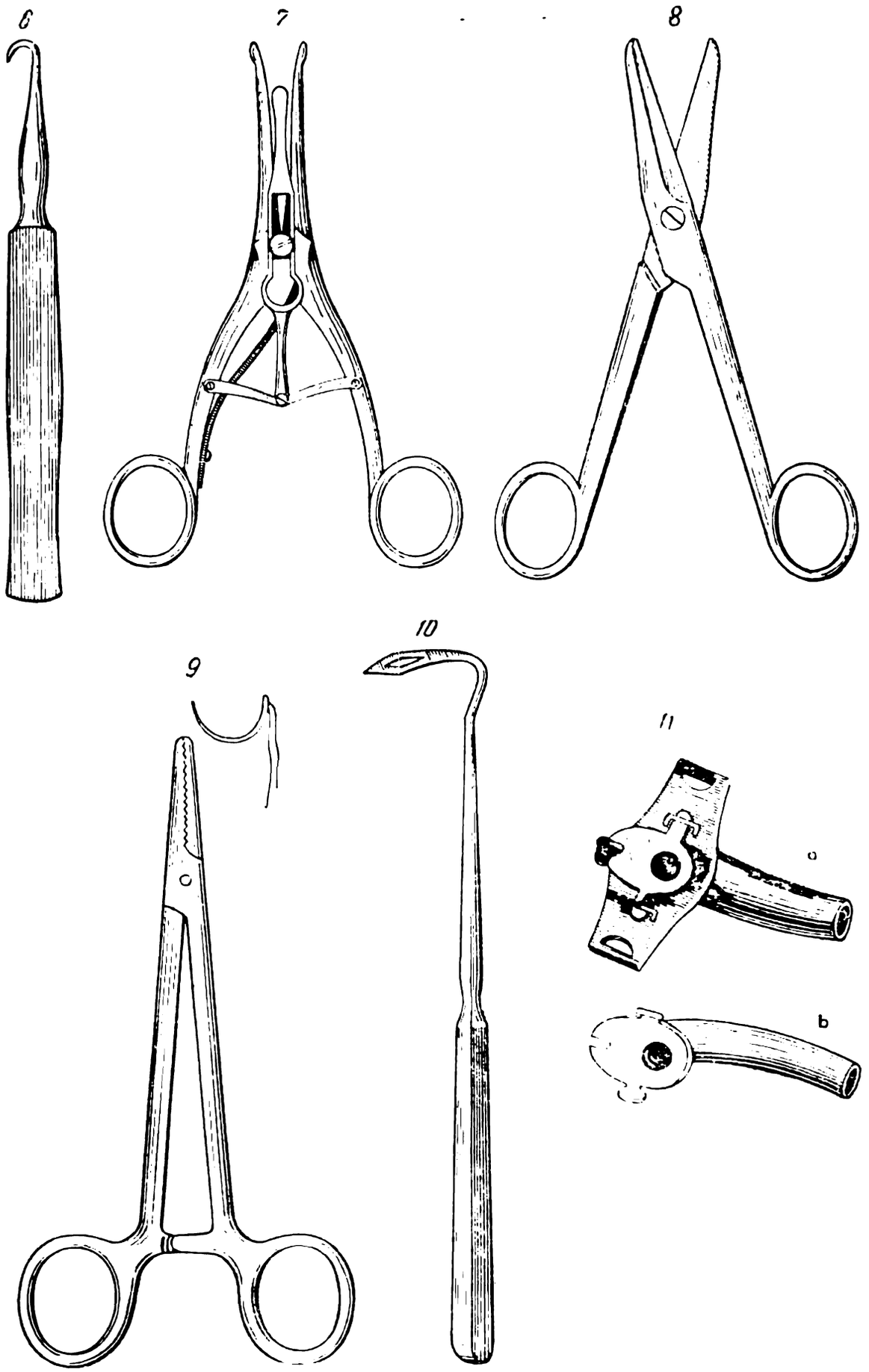


Fig. 113. Set of Instruments for Tracheotomy

(1) syringes with needles for anesthesia; (2) hemostatic forceps; (3) sharp and blunt retractors for widening wound; (4) scalpels; (5) pincers: a—surgical; b—anatomical



(6) sharp hook for fixation of trachea; (7) Trusso's dilator for tracheotomy wound; (8) surgical scissors; (9) needle-holder with needle; (10) ligature needle; (11) tracheotomy tube: a—outer; b—inner

be sufficient time. Before the tracheotomy tube is used it is essential to check that it is firmly fastened to its plate and that the inner tube is in position and can easily be taken out.

Following tracheotomy the patient must be given the most careful attention, especially in the early days of the postoperative period.

It is necessary to ensure that the tracheotomy tube is not choked up with dried mucus or diphtheritic false membranes, which may interfere with respiration.

For this purpose the instillation into the tube of two or three drops of sterile vegetable oil is recommended every two or three hours. In the early postoperative period, the inner tube is taken out two or three times a day, cleansed with a piece of cotton wool wrapped on a flexible threaded probe and sterilised in boiling water.

Following sterilisation in boiling water the tube is dried, lubricated with liquid petrolatum and inserted into the outer tube. In the presence of a viscid secretion from the trachea in the early days of the postoperative period, the inner tube must be cleansed in this manner a few times daily. A method of reducing the drying of the trachea is to humidify the air in the ward by stretching wet bed-sheets across it or to hang a small, wet square of gauze above the tube. The outer tracheotomy tube may be taken out only by a qualified physician.

Measures must be taken to prevent the sputum expectorated by the patient from macerating and irritating the skin of the neck under the tracheotomy tube. For this purpose the skin around the tube is lubricated with petrolatum or Lassar's paste. A square of gauze is placed under the tube without taking it out, and a cover of oil-skin is applied to it and tied up on the far side of the neck. The gauze must be changed as it gets soiled.

The patient's fidgeting, due to violent coughing may cause a loosely fixed tracheotomy tube to fall out. If this happens in the early days, i. e., before formation of a stable granulation channel, the tube may be re-inserted only with the aid of a tracheotomy retractor by loosening the stitches and using hooks to draw apart the lips of the wound.

A too big incision of the trachea and complete stitching of the skin cut may give rise to subcutaneous emphysema,

which is provoked by violent cough. This condition is identified by a markedly swollen neck and characteristic cracking sounds produced by the movement and bursting of air bubbles when the affected areas are being palpated. In such cases, the stitches of the wound must be loosened and the cough allayed by administration of codeine or dionin.

The tracheotomy tube may be removed only after the patency of the larynx has been restored, which is determined by laryngoscopy and functional tests. These are made by plugging the tracheotomy tube. If respiration through the natural air passages then proves to be satisfactory, an attempt to remove the tracheotomy tube may be made and a bandage applied. Meanwhile, a sterile tracheotomy tube and a dilator must always be at hand, so that the wound may be made wider and the tube re-inserted should this become necessary.

FOREIGN BODIES IN THE LARYNX, TRACHEA AND BRONCHI

Various foreign bodies may enter the larynx, trachea and bronchi from the mouth in a sudden inspiration of air, shouting, talking, laughing or eating, or in vomiting. These may be a wide variety of objects, such as sunflower seeds, dentures, drawing-pins, buttons, bones, etc.

The *basic symptoms* of a foreign body in the bronchi or larynx are *repeated paroxysms of a convulsive cough* with attacks of asphyxia. Early physical examination of the lungs usually fails to reveal any changes.

Foreign bodies which have passed through the glottis commonly slip through the trachea and then into one of the principal bronchi. Four-fifths of all foreign bodies lodge in the right bronchus which is wider and deviates less from the vertical direction of the trachea. Some light and smooth foreign bodies jump up and down the trachea with every coughing jerk to produce a characteristic flapping sound when striking the lower surface of the vocal cords. Sometimes, the parents are unable to give an accurate history, and the doctor alone takes the responsibility of deciding whether the foreign body is in the bronchi or in the esophagus.

Foreign bodies in the esophagus commonly cause only pain, a feeling of pressure and dysphagia rather than cough, whereas foreign bodies in the trachea and bronchi cause above all respiratory disorders. Foreign bodies which have stuck in the glottis are responsible for hoarseness or aphonia, which does not occur when they have slipped deeper. Foreign bodies in one of the principal bronchi cause pulmonary atelectasis on the dependent side and emphysematous expansion on the opposite side, detected by percussion and auscultation, as well as by X-ray photography. In the case of metal foreign bodies X-ray examination will aid diagnosis. Bronchoscopy is used to verify the diagnosis and at the same time remove the foreign body. For foreign bodies in the larynx and for mobile foreign bodies in the trachea and bronchi, bronchoscopy may be replaced by an easier method, namely, direct laryngoscopy with the aid of Tikhomirov's or Undrits's spatula which is passed beyond the epiglottis to push the latter outwards (Fig. 114, a, b). The presence of a foreign body in the bronchi for some time will somewhat complicate its detection.

The first requirement is bronchoscopic interference without which the patients often die from various complications, such as secondary pneumonia, lung abscess, etc.

Prophylaxis. This should be in the nature of health education through lectures for parents and personnel of child welfare centres. Emphasis must be laid on the need to keep

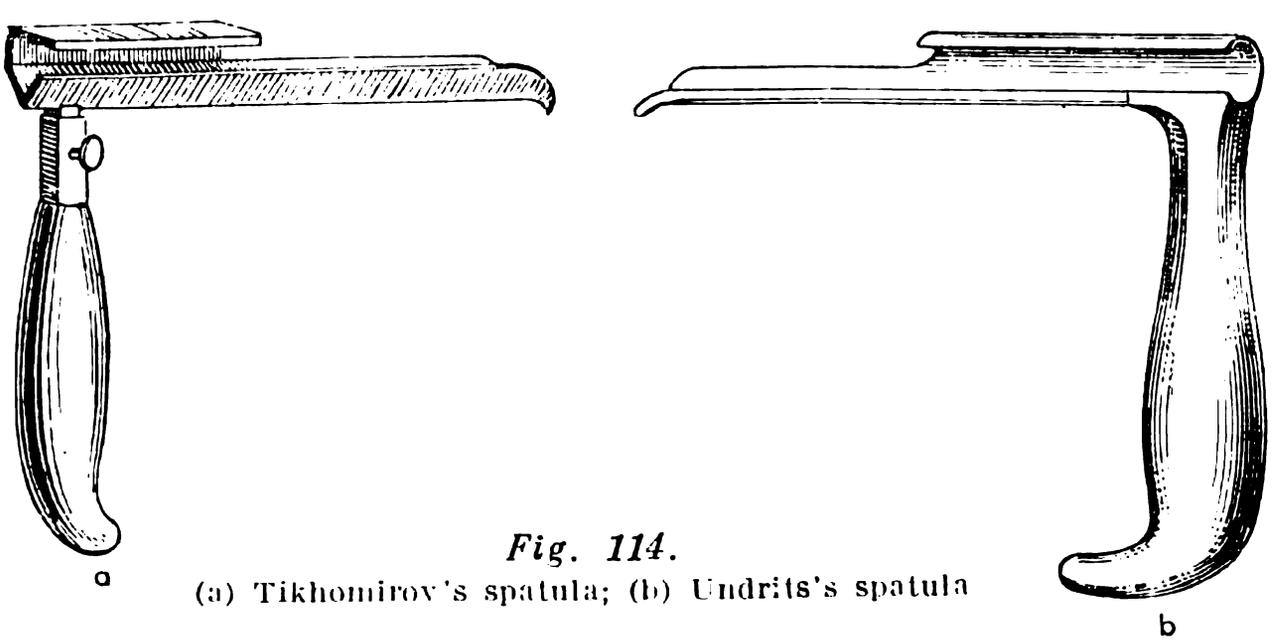


Fig. 114.
(a) Tikhomirov's spatula; (b) Undrits's spatula

a constant watch on children, not to let them play with small objects, like sunflower seeds, peas, cedar nuts, etc., and to wean them from the bad habit of putting objects in the mouth. Children's food must be free of small bones or grains. For example, seeds must be taken out of a water melon or an orange given to a child. Sudden distraction of children at meals is also impermissible.

Children with freely-moving foreign bodies in the larynx are best carried in the sitting posture to avoid displacement of the foreign body and the asphyxia it may cause.

TRAUMATIC LESIONS OF THE UPPER RESPIRATORY TRACT

Injuries of the Nose and Paranasal Sinuses

The protrusion of the nose and fragility of its framework account for frequent nasal injuries even in ordinary conditions. Contusions and gunshot wounds of the nose and paranasal sinuses usually cause bleeding and deformities in some parts of the face. Injuries to the nose often lead to functional disorders due to changes in the nasal passages, and to facial defects due to deformities of the external nose and face. The use of simple methods to correct the defect immediately after trauma may obviate the subsequent need for plastic surgery.

Isolated gunshot wounds of the nose and paranasal sinuses are less common than combined wounds in this region, which penetrate simultaneously into the cranial cavity, eye-socket, pterygopalatine or infratemporal fossae, etc.

There may be contusions, injuries of soft tissue, simple and complicated fractures of the cartilaginous and bony frame of the nose. Facial contusions commonly involve hidden injuries of the nose and paranasal sinuses.

Small fractures of the free margin of nasal bones in such cases may cause no superficial deformities and be detectable only by palpation for a depression in the appropriate place, sometimes by crepitation and, most commonly, by X-ray examination. The most susceptible to injury are the nasal bones whereas traumas of the frontal maxillary processes are less frequent. Injuries to the nasal septum may cause its deviation, dislocation, fissures and fractures. The bony

portion of the nasal septum is less liable to damage in such cases. The elasticity of cartilage accounts for fewer nasal deformities following injuries to the cartilaginous part of the nose. In nasal deformation the cartilaginous part usually follows the bony part, and the correction of the latter is sufficient to restore the former to its normal place. All other kinds of bone fractures of the external nose may be reduced to a few characteristic types.

In the frontal injury to the nasal dorsum, there is a lengthwise fracture of the nasal bones, and the nose is flat owing to the sinking of the dorsum in its bony, and partially in its cartilaginous, portions. There is considerable deformity of the nasal septum or its fracture with formation of a hematoma and rupture of the mucous membrane. The nature of the fracture is determined by means of roentgenography.

In children with poor cohesion of bone sutures, the nasal bones and frontal processes may become separated. The nose will appear even more sunken, and the edges of the frontal processes will be felt on the sides of the sunken part of the nose. Rhinoscopy will reveal dislocation or fracture of the nasal septum.

Lateral dislocations of the nose are particularly frequent. The suture between the nasal bone and the frontal process may fall apart on the affected side with fracture of the frontal process on the opposite side. There is also fracture of the nasal septum and dislocation of the nasal bones from the frontal suture.

Large gunshot wounds or stabs cause abrasions of the nasal and facial skin and shatter the nasal cartilage and lateral bony walls. A bullet fired from a long distance bores a wound passage the exit hole of which is always larger than the entrance.

The position of the bullet passage and holes makes it possible to assess an injury to the nasal bony frame and paranasal sinuses without resort to probing. A still larger trauma with avulsion of soft tissues and bony parts of the face may be caused by mine and shell splinters and bullets fired at close range. In such cases, the whole of the external nose or its separate parts, such as the tip and dorsum, may be disfigured with a certain involvement of the paranasal sinuses.

The symptoms of a nasal injury are: (1) shock, (2) nosebleed ranging from a few drops to a fatal blood loss, (3) pain, swelling and hemorrhages both in the nose and in the adjacent parts of the face, and (4) escape of cerebrospinal fluid through a fissure in the cribriform plate resulting from injury to the roof of the nose.

The skin may be broken in some places while rupture of the nasal mucosa and violent blowing of the nose may give rise to subcutaneous emphysema of the face, eyelids, and other parts of the body. Edema of the skin and swelling of the mucosa quickly increase and interfere with an accurate diagnosis of the fracture.

Diagnosis. In fresh cases of extensive nasal injuries with fractures of the nasal bony frame, the diagnosis is easy, and is largely based on X-ray examination.

Treatment. In closed injuries the first measure is to arrest hemorrhage and reduce the bone splinters as early as possible. Light bleeding from the nose may be controlled by plugging the nose with small tampons soaked in hydrogen peroxide or 2% solution of cocaine with adrenalin. The subsequent tamponade is used not only to arrest hemorrhage but to set bone splinters in their normal place.

Internally, this is achieved by firm packing of the nasal cavity, and externally—by applying compact rolls of gauze to the sides of the nose which are kept in place with strips of adhesive plaster, cleol-covered gauze or with a fairly-tight bandage.

Should severe edema of tissues make exact location difficult, correction of the fracture must be preferably attempted in two or three days' time. In hematoma of the nasal septum a wide incision will be required immediately, to be followed by plugging the nose to prevent abscess formation and destruction of the septal cartilage. An additional X-ray examination is indispensable before taking any measure to reduce bone splinters. Any bleeding from the torn surfaces of an open wound is arrested with a tight dressing, according to first aid rules, or with ligation of the larger vessels in the wound.

Severe hemorrhage from the nasal cavity requires anterior nasal tamponade or posterior tamponade, if the former method proves inadequate.

Severe hemorrhage which does not respond to conservative treatment and endangers life must be checked by ligation of blood-supplying vessels, that is of the external and even the common carotid artery. The presence of external fistulas discharging pus from one or another sinus commonly requires a radical operation.

Injuries to the nose and paranasal sinuses as well as facial wounds heal readily owing to their abundant blood supply.

Upon healing, the wound often leaves a degree of nasal obstruction and nasal deformity which may be corrected by cosmetic surgery.

Injuries of the Pharynx

Wounds of the nasopharynx. Gunshot wounds of the nasopharynx are most commonly combined. The bullet or shell-splinter enters the nasopharynx after passing through the nasal cavity or the antrum of Highmore and sometimes through the eye-socket or the mastoid process. There have been comparatively few cases of damaging missiles penetrating into the nasopharynx through a side of the neck.

Symptoms. These are of general nature and are mostly characterised by accompanying damage, such as concussion of or injury to the brain and injury to the larger vessels. The most conspicuous of the general symptoms is shock. The local symptoms are: (1) bleeding from the nose and mouth as well as the vomiting of blood which has been swallowed in quantity at frequent intervals; (2) obstruction of nasal breathing by clotted blood and sometimes by bone splinters or the damaging object itself clogging the nasal passages; (3) dysphagia due to an injury of the soft palate muscles and superior pharyngeal constrictor; (4) "nasal" voice; (5) pain on turning and inclining the head due to an injury of the upper cervical vertebrae and sometimes to deep-lying cervical muscles.

One of the main complications involves the ear due to the spread of secondary infection from the nasopharynx into the ear through the Eustachian tube. Combined wounds of the nasopharynx involving the sphenoid sinus and backbone are particularly dangerous. The formation of fissures leading

into the cranial cavity may entail severe intracranial complications.

Diagnosis. This is based on the results of physical examination and primarily on those of roentgenography.

Treatment. The first measures in the general treatment are anti-shock therapy, tetanus prevention and compensation for blood loss. Local treatment begins with the arrest of bleeding, often by posterior tamponade. In the most dangerous cases, it may be necessary to tie up the external and sometimes the common carotid artery. Foreign bodies must be extracted through the mouth or nose, and in certain cases through the maxillary sinus. Foreign bodies within easy reach should be removed immediately. A decision to remove a foreign body which is difficult of access can only be made by estimating the danger of its retention and severity of the ensuing functional disturbances in relation to the danger of surgical interference.

Wounds of the oropharynx. Gunshot wounds of the middle part of the pharynx may be either penetrating or blind. The wound channel may pass in various directions. The most common are blind wounds with the wound channel usually passing in the saggital and oblique directions. The damaging object, such as a bullet or shell-splinter, gets caught in the posterior pharyngeal wall or soft tissues of the neck.

Symptoms. The most common are: (1) hemorrhage, (2) dysphagia due to pain or mechanical causes, like a splinter or edema of the soft tissues. Other symptoms are caused by injuries to the adjacent organs and regions, such as the tongue, upper or lower jaw, soft palate and spinal column.

Diagnosis. This usually involves no difficulty in view of the comparatively easy access to the oropharynx for direct examination.

The danger of complications such as aspiration pneumonia and sepsis must be emphasised. Secondary bleeding from large cervical vessels may often threaten the patient's life.

Treatment. This consists in the primary cleansing of the wound and arresting the bleeding often by ligation of the larger vessels. The primary cleansing is done by removing clotted blood, smashed tissue and foreign bodies from the wound and powdering it with white streptocide. Respiratory obstruction caused by an inflammatory edema of the laryn-

geal entrance will require tracheotomy. Clean, external wounds of the neck with little disruption of tissue, either broken or smashed, are sutured layer by layer as follows: the mucosa, the muscles, and, finally, the skin.

Wounds of the laryngopharynx. Wounds of the lower portion of the pharynx are most severe, since they are apt to involve the upper part of the esophagus and the larynx. They are very likely to give such complications, as aspiration pneumonia and suppuration of the mediastinal cellular tissue, called mediastinitis. These wounds also fall into two major groups, the penetrating and the blind.

The symptoms of wounds affecting the laryngopharynx are difficulty in swallowing and sometimes in respiration as well as bleeding from the pharynx. Subcutaneous emphysema, phlegmons, and abscesses on the neck also occur very frequently.

Diagnosis. A direct indication of a penetrating wound in the pharynx or esophagus is escape of saliva or chewed food from the cervical wound. In the absence of this sign, it is rather difficult to detect a penetrating wound in that area. Another essential sign is subcutaneous emphysema, but it may also be the result of a wound in the larynx and trachea without injury to the pharynx or esophagus. Direct examination of wounds in the lower part of the pharynx is very difficult.

The diagnosis of these wounds is greatly facilitated by X-ray examination.

Treatment. Careful and comprehensive surgical treatment of the wound is the primary measure adopted to prevent severe complications. The method of feeding the patient is very important. The most rational is introduction of food into the stomach through a feeding tube which may sometimes be inserted through an external wound. In the treatment of wounds of the pharynx and esophagus the care of the patient is particularly important. Sulfonamides and penicillin are widely used.

Injuries of the Larynx and Trachea

Most injuries to the larynx and trachea come from firearms, whereas wounds made by stabbing, and contusions

are much less common. Gunshot wounds of the larynx and trachea may be of the penetrating, blind and gutter types. Combined wounds with a simultaneous injury to the tongue, pharynx and esophagus are fairly common. The close proximity of large cervical vessels and nerve trunks adds to the danger of these wounds, and is a frequent cause of death.

Symptoms. The most common symptom of injury is a disturbance in phonation varying in degree from simple hoarseness to complete aphonia. Pain on swallowing is always felt in cases of wounds of the larynx and trachea, since deglutition causes excursion of the injured organ. An injury to the upper part of the larynx is marked by choking. A very frequent symptom of injury to the larynx and trachea is difficulty in breathing. Within the early hours of injury respiratory obstruction is commonly caused by blood getting into the air passages or by mechanical obstacles, like splinters of cartilage, scraps of soft tissue and sometimes the damaging object. Later respiratory obstruction is caused by the development of a reactive or inflammatory edema both in the larynx or trachea and in the surrounding soft tissues of the neck. A frequent symptom is cough, sometimes accompanied by secretion from the neck wound and expectoration of foamy blood or saliva and mucus mixed with blood. In the early stages, escape of air from the neck wound may sometimes occur.

Laryngeal and tracheal wounds with a simultaneous injury to the lower part of the pharynx and the upper portion of the esophagus are particularly serious. Apart from hemorrhage and respiratory obstruction, dysphagia is one of the serious complications met with in this condition. A characteristic symptom of such wounds is the entrance of food into the respiratory tract and the escape of swallowed food and fluids through the open wound in the neck. These combined wounds are often aggravated by phlegmons, peripharyngeal and periesophageal abscesses, and sometimes by mediastinitis and sepsis.

These wounds often give rise to subcutaneous emphysema which usually develops when the wound channel is narrow and winding. In such cases, the air pumped by respiration into the wound channel has no outlet and penetrates the hypodermic tissue through cellular interstices. Aspiration

pneumonia is one of the early complications. But the most frequent and formidable complication is stenosis which commonly develops within a few hours of injury and often requires tracheotomy. Among later complications, the worst is secondary bleeding. One of the most serious complications is traumatic perichondritis of the larynx which requires a complicated and prolonged therapy.

Diagnosis. It is relatively easy to identify this condition. In the majority of cases, an injury to the larynx and trachea can be detected by external examination. Escape of foamy blood from the wound on the neck is undisputed evidence of a penetrating wound in the wind-pipe. Cough with expectoration of a blood-stained sputum will also indicate an injury to the larynx or trachea. Timely detection of respiratory difficulties is of great importance. Special attention must be paid to signs of imminent stenosis, such as the indrawing of flaccid parts of the neck and chest, and cyanosis. In blind wounds, X-ray examination helps considerably to determine the exact location of foreign bodies.

Prognosis. The outlook, which is always very serious, depends not only on the size and nature of the injury but also on the accompanying infections and complications. In nearly all fatal cases, the cause of death is bleeding and asphyxia. When the dangers typical of the acute stage have passed, the further dangers of laryngeal and tracheal wounds may arise in later complications, like secondary bleeding, pneumonia, sepsis, and so on.

Treatment. The primary measure is to arrest bleeding and relieve asphyxia. When it is impossible to ligate the bleeding vessel, ligation of cervical vessels should be undertaken.

In progressive respiratory obstruction, tracheotomy is necessary. Today this operation is undertaken only where there is a direct danger of death by suffocation. The primary treatment of the wound is carried out as soon as possible after injury and must be done sparingly.

Foreign bodies which are easy of access, as well as smashed and apparently dying soft tissues are subject to removal. Special care must be paid to the mucous membrane as well as to the perichondrium and laryngeal cartilages.

Damaged cartilages are set back in place and fastened with stitches made through the perichondrium. In many cases a primary suture of the skin wound is possible under penicillin cover. Aftercare consists in watching carefully the condition of the wound and adjoining tissue to prevent purulent complications. In the past few years there has been a sharp fall in the rate of these complications due to the use of *sulfonamides* and *penicillin*. Postoperative care in tracheotomy is in accordance with general rules. The general measures recommended are full quiet, vocal rest, and the use of sedatives, such as morphine or pantopon, to relieve pain and cough, as well as of atropine to diminish the secretion of saliva and mucus. The oral cavity must receive the most thorough care. If the patient is to be moved, he must be accompanied by an experienced medical assistant capable of handling the tracheotomy tube. It must be of sufficient length to prevent it falling out on the journey. The tube may be made longer by the addition of a rubber tube of appropriate diameter.

OCCUPATIONAL DISEASES OF THE UPPER RESPIRATORY TRACT

The principal occupational hazards for the upper respiratory tract are these: (1) dust, (2) vapours and gases, and (3) unfavourable weather conditions, such as excessive dryness or humidity of the air, sharp temperature changes, draughts, and so on.

The favourite site of affection is the nasal cavity, which is the opening portion of the upper respiratory tract, and is, therefore, most susceptible to harmful influences. Second in frequency are lesions of the distal sections of the respiratory tract. Lesions of the nasopharynx may entail diseases of the Eustachian tubes and middle ear.

Dust. Of all occupational factors dust has the most pernicious effect on the upper respiratory tract.

Dust may be of inorganic or organic character. Inorganic dust may in its turn be mineral or metallic, whereas organic dust may owe its origin to vegetables and animals. Dust has a mechanical or chemical effect depending on its solubility and absorption. The common types of industrial

dust are met with in handling iron, brass, copper, lead, silicate, cement, lime, etc. Vegetable dust may come from flour, wood, tobacco, and cotton, while animal dust forms in processing wool, hairs, skins, etc.

These dusts inflame the mucosa of the nose, pharynx and larynx by mechanical irritation. The degree and location of affection depends on the physical properties of the inhaled dust particles, i.e., their weight, size, form, speed of flight, etc. The heavier particles of metallic and mineral dust primarily invade the anterior parts of the nose, whereas the lighter particles, those of hemp, wool, etc., affect the deeper portions of the respiratory tract. Dust particles flying at a high speed, like in the processing of metal, porcelain, and other materials, have a greater traumatic action. The direct traumatic effect produced on the mucous membrane by soft particles of round shape, like those of chalk, gypsum and flour dust, is not particularly severe. These dusts, however, quickly obstruct the outlet ducts of the mucous glands, drying up the mucosa so that atrophic processes enter into the picture.

The first symptoms of acute inflammation of the mucosa are tickling in the nose, sneezing and a fairly copious nasal discharge. The swollen nasal mucosa interferes with nasal respiration and makes the patient breathe through the mouth and thus inhale a still greater amount of dust. Mouth breathing causes irritation in the pharynx with a feeling of dryness and itching in the throat accompanied by a constant temptation to clear the throat and a harsh cough. Catarrhal or suppurative otitis is caused when the nasopharynx and Eustachian tube become involved in the process.

Very often catarrhal inflammation leads to atrophy of the mucosa with subsequent degeneration of the glandular system and replacement of the columnar ciliated epithelium by the squamous type. Nearly all of the dust-provoked respiratory diseases belong to catarrhal and atrophic processes. Apart from inflammations in the nasal cavity, workers in some dusty industries, flour mills, cement plants, etc., have been observed to develop anterior rhinitis sicca, which may be accompanied by erosions and ulcers, recurrent nosebleed and perforation of the nasal septum. The latter commonly occurs in the anterior cartilaginous part and

usually leaves the bony portion intact. It is most frequently found among chromium industry workers, and less commonly among those engaged in cement and caustic acid production.

The *chemical effect* of certain kinds of dust is always combined with their traumatic action. The action of a strong saline solution will bring to the fore symptoms of a catarrhal inflammation often followed by tissue hypertrophy. A chemical burn is often followed by scarring, shrivelling and atrophy of tissues. A specific effect on the mucous membranes is produced by the salts and vapours of chromic acids which cause limited necrosis not only in the nasal mucosa but in the septal cartilage with its subsequent perforation.

In the majority of cases, the harmful influence of *vapours* and *gases* is due to their toxicity, and in addition to local symptoms, they often produce general intoxication. The extent of affection of the mucosa of the upper respiratory tract depends, above all, on the solubility of a particular gas in water. This explains, for example, why ammonia particularly affects the nose and pharynx, while phosgene primarily attacks the pulmonary alveoli. Poisoning with certain gases serves to diminish the resistance of the mucosa to some kinds of pathogenic germs. Thus, hydrogen sulfide and sulfurous gas poisoning reduces resistance to streptococci and staphylococci, which in turn, contributes to inflammation developing in the mucosa of the upper respiratory tract. Sulfurous vapours (anhydrites) cause irritation in all the mucous membranes of the upper respiratory tract. In these cases, the clinical symptoms are those of inflammation, ulceration and necrosis of the mucosa.

Acute gas poisoning gives symptoms of acute rhinitis and acute laryngitis, whereas chronic gas poisoning produces a chronic catarrhal inflammation of the mucosa. The lesion often involves the receptor of the olfactory analysor, the sense of smell being lost in large measure or altogether.

The *vapours of acids*, such as sulfuric, hydrochloric, nitric, and other acids, irritate the mucous membranes and provoke inflammations in the mucosa of the upper respiratory tract, which may be acute or chronic depending on the duration of harmful influence. The action of acids essentially consists in the formation of albuminates by combination with cellular proteins.

Alkalies possess milder cauterising properties but they have a marked diluting effect on protoplasm. The inflammatory reaction is somewhat milder, yet it tends to extend and often ends in a considerable destruction of tissue.

A number of volatile oils, oil products and turpentine oil cause a sharp irritation in the mucosa and its subsequent gradual atrophy. At an early stage, the mucosa is hypersensitive and irritated. This is followed by full loss of sensitivity and complete or partial loss of the sense of smell. In a number of industries, combined influences of vapours, harmful gases and dust are found.

One of the first symptoms of affection of the mucosa, for instance in the nose, is copious liquid secretion and edema. The simultaneous influence of other harmful factors, such as dust, gas and infection, causes marked inflammation in the mucosa of the upper respiratory tract. In the absence of prophylactics, atrophy of the mucosa develops in those who work in hot, dry and dusty surroundings, namely, stokers, foundry-workers and furnacemen.

Prophylaxis of occupational diseases of the upper respiratory tract is based on collective and individual measures. All such measures of social importance have been enforced by Soviet legislation and the appropriate labour safety regulations. Lately, special emphasis has been laid on medical advice concerning occupation and the prophylactic treatment of the upper respiratory tract. This advice, based on medical examination, enables a newly-employed young worker to be appointed to a job that is best suited to his mental and physical abilities and helps the administration to select for especially harmful jobs men with the best physical fitness and capacity for adaptation.

For example, a juvenile with a far-advanced atrophic rhinitis working in fairly dusty surroundings will prove more susceptible to dust than his healthy counterpart. Another example: a juvenile with adenoids and chronic rhinitis must undergo prophylactic treatment of the upper air passages, that is have his adenoids removed and rhinitis cured before he can be allowed to take up his duties.

This was one of a long list of examples which prove beyond doubt that medical advice about occupation and prophy-

lactic measures are quite necessary before appointment to a regular job.

Individual prophylaxis consists of lubricating the nasal cavity with various fatty substances (in chromium production) or by using different oil drops for the nose as well as of regular douches of the nose, pharynx and larynx in dusty industries and by inhalation. Inhalation may have a decisive effect if carried out immediately upon completion of work in dusty surroundings. There are special masks or respirators available to prevent the inhalation of noxious substances into the upper air passages.

Collective prophylaxis essentially consists in measures to improve the manufacturing process, and so to a great extent eliminate the root causes of harmful industrial factors: dust, vapour, gas, etc. For this purpose improved ventilation systems have been introduced on a particularly wide scale.

These measures are taken at all Soviet plants and factories and occupy an important place in new industrial projects.

Other methods of prophylaxis that are regarded as exceedingly important for health promotion in the U.S.S.R. are regular holidays which are longer on jobs with health hazards, advancement of public welfare and cultural standards, holidays in rest-homes and sanatoria, free medical aid, alcoholism control, and so on.

DISEASES OF THE ESOPHAGUS

ANATOMY OF THE ESOPHAGUS

The esophagus is a muscular tube of about 25 cm in length which extends from the lower portion of the pharynx, on a level with the upper border of the cricoid cartilage, passes downwards through the thoracic cavity and pierces the diaphragm before it turns into the stomach.

The esophagus consists of cervical, thoracic, and abdominal parts. The esophageal walls consist of three layers: (1) the mucosa covered with squamous epithelium, (2) the muscular layer consisting of lengthwise and crosswise muscle fibres, and (3) the external layer made up of dense and fibrous connective tissue intertwined with a certain number of elastic fibres.

From the practical point of view it is important to remember that the lumen of the esophagus is not uniform all along its length, but has three structural narrowings, the first—at the beginning of the esophagus, the second—at the point of intersection with the left bronchus, and the third—at the stomach entrance. Foreign bodies tend to lodge in these narrow spaces. In adults, the distance from the upper edge of the teeth to the first narrowing is 15 to 17 cm, to the second narrowing—22 to 25 cm, and to the stomach entrance—40 to 42 cm.

METHODS OF EXAMINING THE ESOPHAGUS

Prior to actual examination, it is important to establish in detail the patient's complaints of dysphagia. The most common examination is roentgenoscopy and roentgenography of the esophagus through the contrast medium. The exploration of the esophagus, treatment of its diseases and extraction of foreign bodies are carried out by means of endoscopy, more specifically esophagoscopy, which is a highly valuable diagnostic and therapeutical method.

BURNS AND STRICTURES OF THE ESOPHAGUS

Burns of the esophagus are caused by hot food or acids and alkaline caustics whose destructive effect may be quite severe. The catarrhal, fibrinous, or necrotic changes which may occur in the esophagus will depend upon the properties and concentration of the chemical swallowed. Necrosis of the esophageal walls may occasionally be so deep as to invade the muscular lining and threaten perforation of the entire esophageal wall and may often cause mediastinitis. In milder cases, subsequent cicatrisation may entail strictures of the esophagus, which can also develop as a result of previous inflammations or ulcerations of its walls. The lumen of the esophagus may become narrower due to outside tumours pressing on its walls or be obstructed by developing internal tumours which may block it altogether.

The patients complain of dysphagia and sometimes of full throat obstruction; in milder cases, it is only difficult to swallow solid food while liquid food can be taken easily. These patients rapidly lose weight.

The *first aid* rendered immediately after the swallowing of acids or alkalies consists in the earliest possible and careful lavage of the stomach, at least within the first six hours and neutralisation of the toxic matter.

In **acid poisoning** the stomach is washed out with warm water or a warm magnesium oxide solution. Milk of lime and mucilaginous decoctions must be given orally, and it is advisable to swallow pieces of ice.

In **alkaline poisoning** the stomach is washed out with water containing acetic acid, and the patient is instructed to take a 1% solution of tartaric, acetic, or citric acid, drink a lot of milk, mucilaginous decoctions, oil emulsions, and swallow pieces of ice.

At the same time, anti-shock measures are essential, and a hypodermic injection of a 1 ml dose of 2% pantopon or omnopon solutions or a 1 ml dose of 1% morphine solution is given. If indicated, camphor, caffeine, and a physiological solution are prescribed for injection under the skin.

To reduce the inflammatory reaction and prevent the grave complications which so often occur after burns, mas-

sive penicillin therapy is given on the very first day, and should be continued for another six or nine days.

Treatment. A protective diet of milk, cream, whipped raw eggs, mucilaginous broths, etc., is given during the first week after the accident. Vegetable oils, such as olive or sunflower oils, given in a tablespoonful dose five or six times a day will have a soothing effect on the scalded mucosa. The patient is advised to drink plenty of liquid, and in the case of dysphagia the nutritive enema is used, as well as infusions of physiological and 5% glucose solutions, and if necessary a blood transfusion may also be given. When the inflammatory symptoms subside and if there are no complications, the patient should be soon put on his usual diet avoiding the intake of roughage. Chemical burns of the esophagus are successfully treated with "early" bouginage undertaken by the doctor after the acute symptoms have subsided, from the fifth or sixth day in light cases, and from the eighth to twelfth day in cases of moderate severity. This procedure must be continued for two or three months. In chronic cases, where a stubborn cicatricial stricture has already formed, the "late" bouginage is used, which is very difficult, less effective, and is often followed by recurrent strictures.

The bougie is not boiled but is rinsed in warm water and wiped dry with a piece of cotton wool soaked in a disinfectant, usually 1% chloramine solution.

The *prophylaxis* of cicatricial strictures of the esophagus following caustic burns consists essentially in an efficient and timely emergency aid as well as in correct treatment within the first days of the accident. The serious effects of caustic burns should be made widely known through lectures on hygiene and sanitation, posters, etc. At the same time the sanitary authorities must see to it that caustic preparations, such as caustic soda or acetic acid, are sold in special packages or bottles labelled "poison".

FOREIGN BODIES OF THE ESOPHAGUS

A variety of objects are apt to get caught in the esophagus, such as fish bones, coins, buttons, nails, and so on. They usually lodge in structural narrowings, although sharp

and rough objects may become stuck in any part of the esophagus. The majority, in fact 75 per cent, of all foreign bodies in the esophagus lodge in its upper third part. The symptoms of a foreign body are difficulty in swallowing, dull pain and pressure in the chest or along the backbone. Sharp foreign bodies sticking in the esophageal walls may sometimes cause perforation, which is accompanied by a shooting pain and symptoms of phlegmonous periesophagitis or mediastinitis. This occasionally proves fatal.

The diagnosis rests on the detailed history obtained from the patient, X-ray examination with the contrast medium, and sometimes roentgenography. When indicated, esophagoscopy must be undertaken as a diagnostic as well as a therapeutical measure to remove the foreign body under the guidance of vision. Probing and random attempts to extract the foreign body with various instruments, coin extractors or forceps, are strictly forbidden.

If the esophageal passage is swollen and tender following an unsuccessful attempt to extract the foreign body, the best procedure is to give the patient penicillin in large doses and wait for two or three days until the symptoms of traumatic esophagitis have subsided.

In the event of scratches on the esophageal walls which may cause a spasm and dull pain in the esophageal passage, a bland diet of liquids and gruel and intramuscular penicillin injections are recommended for a few days, as well as atropine and atropine drugs to relieve the spasm of the esophageal smooth muscles. Where it is impossible to extract the foreign body with an esophagoscope, esophagotomy will be required.

CANCER OF THE ESOPHAGUS

Cancer of the esophagus accounts for a fairly large proportion of the total number of malignant tumours, and is commonly encountered in males after the age of 40 or 50.

Dysphagia in an elderly person must always be checked for cancer. At an early stage of esophageal cancer dysphagia is not infrequently a sign of the accompanying spasm of the esophagus and may grow worse or better according to the kind of food being eaten and the state of the nervous system. Pain may be caused by ulceration or spasm of the inflamed walls

of the esophagus. At first, the symptoms may be those of a feeling of discomfort or pressure behind the sternum on swallowing, while later they may turn into a sort of pulling and rending pain radiating to the neck, the area between the shoulder-blades or epigastric region. Moreover, these symptoms depend in some measure on the site of the tumour. There is often vomiting and belching at meals, increased salivation and bad breath. Nevertheless, the final diagnosis rests on X-ray examination and esophagoscopy.

Treatment. Nowadays, the treatment of esophageal cancer, particularly in its early stages, is not as hopeless as in the past owing to the great advances made in thoracic surgery. Radiotherapy may also be used if indicated. In far-advanced cases with marked difficulty in deglutition gastrotomy is used to delay the fatal outcome.

Prophylaxis. There is evidence that a constant abuse of liquors, tobacco, and hot food has a bearing on the rate of esophageal cancer. This fact must be emphasised in any lecture or discussion on the subject of health promotion.

HEALTH EDUCATION

Apart from their regular duties, junior medical personnel are responsible for organising a health education campaign in order to raise the sanitary standards of the community and disseminate knowledge concerning collective and individual measures required to prevent infectious, parasitic and epidemic diseases, as well as on the elements of personal and industrial hygiene.

The emphasis on prophylactics so characteristic of Soviet medicine is more evident in otorhinolaryngology than in any other of its branches. Indeed, no matter which department of otorhinolaryngology is being considered, the tremendous role of prophylaxis and treatment of E.N.T. diseases in the prevention of constitutional disturbance always stands out clearly. For example, the timely treatment of acute and chronic tonsillitis serves to ward off such grave diseases as rheumatism, nephritis, infectious polyarthritis, etc. The timely cure of nasal and nasopharyngeal diseases prevents repeated catarrhs of the upper respiratory tract, diseases of the middle ear, and so on, while the treatment of middle otitis averts the formidable danger of intracranial complications, like meningitis, sepsis, cerebral abscess, etc. A large-scale organisation of lectures and discussions on otorhinolaryngology, distribution of pamphlets and leaflets on the same subject will undoubtedly help in the early detection of E.N.T. diseases, as well as in their timely treatment, and will, thereby, serve to prevent many of their complications. It is the duty of junior medical personnel to regard health education as an integral part of their regular clinico-prophylactic work.

Below we give a brief list of recommended topics for lectures and discussions on health education.

1. Tonsillitis and Its Control.
2. Nasal Obstruction in Children and Its Consequences.
3. Prevention of Hearing Defects.
4. Prevention of Intracranial Complications of Suppurative Otitis Media.
5. Nasal Bleeding and Its Arrest.
6. Advice to Patients with Chronic Ear Pus Discharge.
7. Chronic Tonsillitis.
8. Hardening of the Body as a Prophylactic Measure against Diseases of the Upper Respiratory Tract.
9. The Cause and Treatment of Chronic Rhinitis.
10. The Harmful Influence of Smoking on the Senses of Smell, Taste and Hearing.

The present text-book can supply all the necessary information for lectures and discussions on E.N.T. diseases. The lecture or discussion should take between 20 or 30 minutes and be delivered in simple language understandable to all those present. Examples taken from everyday life will make the lecture or discussion more interesting.

Apart from lectures and discussions delivered to large audiences, any individual suffering from an ear, nose or throat disease should be given a detailed explanation of its nature and instructed how to prevent its recurrence. It is equally important to point out the complications likely to follow the patient's neglect of medical prescriptions.

The following is a model plan for lectures or discussions on two different subjects.

First subject: "Tonsillitis and Its Control"

The lecture should be started with a brief outline of the anatomy and physiology of the faucial tonsils before describing the changes seen in the fauces in catarrhal, lacunar and follicular tonsillitis. It must be emphasised that this disease is a constitutional disturbance requiring rest in bed and hygienic habits. Since tonsillitis may be due to diphtheria or scarlet fever, it is highly important to consult a doctor as early as possible, so that the patient may be taken to a hospital for infectious diseases in good time. The gravest complications of tonsillitis such as rheumatism, nephritis, etc., must also be pointed out. However, the highlight of the lecture must be measures of tonsillitis prevention, such

as the provision of a separate bed and eating utensils for a tonsillitis patient, so as to avoid transmission of the infection to other people. For the same reason, the patient's room must be ventilated and cleaned with a wet rag. It must be stressed that nasal obstruction due to adenoids, chronic rhinitis or chronic sinusitis may be the cause of repeated tonsillitis. Dental caries is also one of the predisposing causes. As tonsillitis is often preceded by a chill, special attention must be given to body hardening. Detailed explanations on how to take air and sun baths, do one's daily dozen, sponge down one's body, etc., should be given.

The part played by a sensible division of work and rest, a good diet, physical culture and sport in the maintenance of good health must also be stressed.

Second subject: "Nasal Obstruction in Children and Its Consequences"

After showing the importance of normal nasal breathing for the body and giving a concise description of the anatomy and physiology of the nose, it must be pointed out that the most frequent causes of respiratory obstruction are adenoid hyperplasias in children, chronic rhinitis and chronic inflammations of paranasal sinuses, while tumours of the nose, which are confined to adults, less commonly obstruct its air passages. When outlining the symptoms of this condition a brief commentary must be given on the mode of treatment. It is then underlined that nasal obstruction causes repeated affections of the air passages lying next to the nose as well as otitis media and a loss of hearing. Mention must be made of the negative effect of adenoids on the child's development shown in skeletal deformities, mental retardation, etc.

Every junior medical worker must conduct his part of a health education campaign aided by various organisations and in close co-operation with local Red Cross or Red Crescent Society workers and drawing on the support of other public organisations. Every effort must be made to see to it that everybody understands the importance of sanitation and of abolishing occupational hazards as well as observing labour safety regulations.

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